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A STUDY OF THE RELATIONSHIP OF CONVULSIONS IN INFANCY AND CHILDHOOD TO EPILEPSY*

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About twenty or twenty-five years ago I was much interested in the discussion which was then going on as to the connection between convulsions in early life and epilepsy. I felt that at that time I had no right to an opinion of my own, but resolved to keep the matter in mind and to see later if I could draw any conclusions from my own experience. I have, therefore, looked up, as far as possible, the condition of the babies and children with convulsions whom I have seen in consultation and in private practice. I have included only those cases in which the convulsions were the primary cause for my attendance and have excluded all of those cases in which there were any evidences of acute or chronic cerebral disease. I have had two objects in mind in this study: first, to determine what proportion of the children, otherwise apparently normal, having convulsions have epilepsy or develop it later, and, second, to find out, if possible, whether there is anything in the history or in the manner of the development of the convulsions to show whether or not they are manifestations of epilepsy, or whether they will be followed by or develop into epilepsy later.

I have been able to obtain satisfactory reports regarding 107 children. The time elapsed has varied between two and twenty years. In order to study these cases better and to avoid confusion, I have divided them into four classes: first, those cases in which the convulsions were associated with evidences of spasmophilia; second, those cases in which the convulsions occurred in the course of whooping cough; third, those cases in which there was a single convulsion or a series of convulsions at the onset of some acute disease or with an attack of acute indigestion; fourth, those cases in which there had been repeated convulsions during a considerable period or in which there had been repeated attacks suggesting petit mal. I shall take up each of these classes separately.

* Read before the Thirty-First Annual Meeting of the American Pediatric Society, held at Atlantic City, June 16-18, 1919.

1. *Cases Associated with Evidences of Spasmophilia.*—There were ten cases in this class, all occurring in infants. In one instance the convulsions had lasted only twenty-four hours. In the other cases there had been many attacks during two weeks, three weeks, three weeks, one month, two months, three months, three months, seven months and nine months, respectively. Four of these babies had no more convulsions, and one had only one more. Two continued to have convulsions for a week, and one of them intermittently for a year. In another, the attacks persisted for two years. It was finally decided, however, that these attacks had changed in character, and that the child induced them voluntarily in order to obtain its wishes. They stopped at once, never to return, when a glass of cold water was thrown in the child's face at the beginning of an attack. In another instance, the attacks persisted for two years and then ceased. This child, however, became feebleminded. It is probable, therefore, that in this instance the convulsions were not a manifestation of spasmophilia, but were due to some other cause. This supposition is the more probable, because the first attacks appeared coincidently with an attack of influenza and otitis media. She died of infantile paralysis when 9 years of age. All the other children are now alive and well. The time elapsed since these children were seen has been four, five, seven and one-half, eight, nine and one-half, eleven and one-half, twelve and one-half, twelve and one-half, fourteen and one-half and twenty years, respectively.

It would seem, therefore, that the convulsions which occur in babies with spasmophilia, and which are presumably a manifestation of spasmophilia, are not likely to lead to the development of epilepsy later.

2. *Cases in Which the Convulsions Occurred in the Course of Whooping Cough.*—There were three cases in this class. In one case, a child 6 years of age, there was only one convulsion. She was unconscious, however, for several days. She has been and is now well, three years later. Another, a baby four months of age, had several convulsions in twelve hours. The child has been and is now well, eleven and one-half years later. The third patient, a baby, sixteen months of age, had two convulsions in four days. She developed epilepsy two years later and still has attacks, sixteen and one-half years after the attack of whooping cough. She is normal mentally and physically.

These cases are too few in number to warrant any definite conclusions. They show, however, that convulsions in whooping cough should be regarded seriously, and that they may lead to the development of epilepsy later. They also seem to show that no conclusions

TABLE 1.—Cases in Which There Had Been Repeated Convulsions During a Considerable Period or in Which There Had Been Repeated Attacks Suggesting Petit Mal

A. EPILEPTICS

No.	Age When Seen	Age at Onset	Duration When Seen	Nature of Attacks	Apparent Etiology When Seen	Diagnosis When Seen	Labor	Possible Etiology	Results
1	8 yrs.	5 yrs.	3 yrs.	Petit mal	None	Epilepsy or hysteria	Normal	None	Grand mal, feeble-minded
2	10 yrs.	Infancy and again at 10 yrs.	10 yrs.	General	Indigestion	Reflex convulsions	Normal	None	Normal for 7 yrs., then grand mal
3	3½ yrs.	3½ yrs.	5 wks.	General	None	Reflex convulsions or epilepsy	Forceps	None	Attacks continued, grand mal
4	3¾ yrs.	17 mos.	2 yrs.+	General	Indigestion	"Reflex convulsions" or epilepsy	Normal	None	Normal for 6 yrs., then petit mal
5	5 yrs.	5 mos.	One convulsion; petit mal beginning one month before seen	Petit mal	None	Petit mal	Breech, hard to resuscitate	Injury at birth or first convulsion	Grand mal
6	10 yrs.	1 yr.	9 yrs.	General	None	Epilepsy	Normal	None	Grand mal
7	7½ yrs.	6 mos.	One convulsion; petit mal beginning six months before seen	Petit mal	Injury from first convulsion?	"Petit mal" or reflex convulsions	Normal	Injury from first convulsion	Grand mal, feeble-minded
8	18 mos.	13 mos.	5 mos.	General	Indigestion	"Reflex convulsions" or epilepsy	Normal	None	Grand mal, feeble-minded
9	5 yrs.	2 yrs.	3 yrs.	General	Injury to head from fall	"Epilepsy" or reflex convulsions from indigestion	?	Injury from fall	Grand mal, feeble-minded
10	4 yrs.	3½ yrs.	6 mos.	General	Indigestion	Epilepsy	Normal	None	Petit mal
11	5 yrs.	1½ yrs.	3½ yrs.	General and petit mal	Indigestion	"Reflex convulsions" or epilepsy	Hard	None or hard labor	Petit mal
12	12 yrs.	11 yrs.	1 yr.	General	Injury to head from kick of horse or nervousness	"Reflex convulsions" or epilepsy	?	Injury to head	Grand mal
13	10 mos.	5 mos.	5 mos.	General	Indigestion	"Reflex convulsions" or epilepsy	Hard	Hard labor	Grand mal, feeble-minded
14	5 yrs.	1 yr.	4 yrs.	General	None	Epilepsy	Normal	None	Grand mal
15	2½ yrs.	1½ yrs.	1 yr.	Petit mal	Hard labor	Petit mal	Hard	Hard labor	Grand mal
16	7 yrs.	1½ yrs.	Two convulsions at 18 months; petit mal beginning one year before seen	Petit mal	Hard labor or first convulsion	Petit mal	Hard	Hard labor or first convulsion	Grand mal
17	7½ yrs.	Birth	Two convulsions in first 24 hours, two in next four days; no more until one week before seen; from fright	General	Fright	Eclampsia, not epilepsy	Hard	Hard labor or first convulsion	Grand mal

TABLE 1.—Cases in Which There Had Been Repeated Convulsions During a Considerable Period or in Which There Had Been Repeated Attacks Suggesting Petit Mal—*Continued*

B. NOT EPILEPTICS

No.	Age When Seen	Age at Onset	Duration When Seen	Nature of Attacks	Apparent Etiology When Seen	Diagnosis When Seen	Labor	Time Since Seen	Time Since Last Convulsion	Remarks
18	3 yrs.	2 yrs.	1 yr.	General, 5; fainting attacks also	Indigestion	Reflex convulsions	Normal	4 yrs.	2 yrs.	Two in next 2 years, from indigestion
19	6 mos.	Birth	6 mos.	General, many	Otitis media, indigestion	Reflex convulsions	Forceps	3 yrs.	2 yrs.	Continued for 1 year, until cut last tooth
20	3 yrs.	1½ yrs.	1½ yrs.	General, 3	Indigestion	Reflex convulsions	Normal	3 yrs.	3 yrs.	No more convulsions
21	6 yrs.	2 yrs.	4 yrs.	Petit mal, 7	Indigestion	"Reflex convulsions" or epilepsy	Forceps	3½ yrs.	3½ yrs.	One slight attack soon after
22	8 mos.	6 mos.	2 mos.	General, 2 sets	Indigestion	Reflex convulsions	Forceps	3½ yrs.	3½ yrs.	No more convulsions
23	2 mos.	6 wks.	2 wks.	Several severe and many slight	Indigestion	Reflex convulsions	Cesarian	4 yrs.	4 yrs.	No more convulsions
24	7½ yrs.	7 yrs.	6 mos.	General, 6 in last 3 weeks, slight attacks of stiffness in leg for 6 months	Indigestion	"Epilepsy," tumor or reflex convulsions	Normal	9½ yrs.	4½ yrs.	Gradually diminished in frequency and ceased
25	9 mos.	8 mos.	1 mo.	General, 5	Circumcision, 1; indigestion, 4	Reflex convulsions	Forceps	4½ yrs.	4½ yrs.	Stopped after a few days
26	2½ yrs.	1½ yrs.	1 yr.	General, 5	Indigestion and onset of acute disease	Reflex convulsions	Forceps	5½ yrs.	4½ yrs.	One or two in next year from indigestion
27	7 yrs.	6½ yrs.	6 mos.	General, 2	Overfatigue and indigestion	"Reflex convulsions" or epilepsy	Forceps	5½ yrs.	5 yrs.	One or two in next 6 months
28	20 mos.	12 mos.	8 mos.	General, 4	Fright or crying	"Convulsions from nervousness" or epilepsy	Forceps	5½ yrs.	5½ yrs.	Two from pertussis soon after
29	3 yrs.	2½ yrs.	6 mos.	Petit mal	Began with an acute illness	"Functional" or epilepsy	Normal	6½ yrs.	5½ yrs.	Gradually diminished in frequency and ceased
30	3 yrs.	2½ yrs.	6 mos.	Petit mal	Indigestion	"Reflex convulsions" or epilepsy	Normal	5½ yrs.	5½ yrs.	Attacks soon ceased
31	9 mos.	8 mos.	1 mo.	General, 1; then many in 24 hrs. 3 weeks later	Indigestion	Reflex convulsions	Forceps	5½ yrs.	5½ yrs.	No more convulsions
32	4 yrs.	2½ yrs.	1½ yrs.	General, 12 in 18 months	Indigestion	"Reflex convulsions" or epilepsy	Normal	7 yrs.	6 yrs.	Occasionally for 1 year

33	2½ yrs.	1 yr.	1½ yrs.	General, 3	Indigestion	Reflex convulsions	Normal	7 yrs.	7 yrs.	No more convulsions
34	9 mos.	Birth	9 mos.	Petit mal	Adenoids or hard labor	"Reflex convulsions" or epilepsy	Hard	10 yrs.	7 yrs.	Occasional attack until adenoids removed
35	2½ yrs.	2½ yrs.	6 wks.	General, 6	Indigestion and otitis media	Reflex convulsions	Normal	7½ yrs.	7½ yrs.	No more convulsions
36	10 yrs.	9 yrs.	1 yr.	General, several; petit mal, many	None	Epilepsy	Forceps	9 yrs.	8½ yrs.	Attacks soon ceased
37	6 mos.	Birth	6 mos.	General, few; many slight attacks of tremor	Injury to mother during pregnancy	"Epilepsy" or reflex convulsions	Forceps	9 yrs.	9 yrs.	No more attacks
38	22 mos.	2 mos.	20 mos.	General	Indigestion	Reflex convulsions	?	9 yrs.	9 yrs.	No more convulsions
39	3 mos.	2 mos.	1 mo.	General, 3	Indigestion	Reflex convulsions	Normal	9 yrs.	9 yrs.	No more convulsions
40	6 yrs.	3 yrs.	3 yrs.	Petit mal	None	Epilepsy	Normal	9 yrs.	9 yrs.	No more convulsions
41	3½ yrs.	2¾ yrs.	9 mos.	General, 4	Indigestion	Reflex convulsions	Normal	9½ yrs.	9½ yrs.	No more convulsions
42	8 yrs.	1 yr.	7 yrs.	General, few	Indigestion	Reflex convulsions	?	10 yrs.	10 yrs.	No omer convulsions
43	27 mos.	5 mos.	22 mos.	General, many	Indigestion	Reflex convulsions	?	11½ yrs.	11½ yrs.	No more convulsions
44	7 mos.	2 mos.	5 mos.	General, many	Indigestion	Reflex convulsions	Forceps	11½ yrs.	11½ yrs.	No more convulsions
45	11 mos.	9 mos.	2 mos.	General, many	None	"Reflex convulsions" or epilepsy	Normal	13½ yrs.	11½ yrs.	Attacks diminished in frequency for 2 years, then ceased
46	21 mos.	18 mos.	3 mos.	General, 2	Bronchitis, indigestion	"Reflex convulsions" or epilepsy	Normal	12 yrs.	12 yrs.	No more convulsions
47	4 mos.	3 mos.	1 mo.	General, many	Indigestion	Reflex convulsions	Normal	13½ yrs.	12 yrs.	One attack 1½ years later
48	2 yrs.	18 mos.	6 mos.	General, 2	Indigestion	"Reflex convulsions" or epilepsy				
49	18 mos.	12 mos.	6 mos.	General, 5	Constipation	Reflex convulsions	Normal	12 yrs.	12 yrs.	No more convulsions
50	6 mos.	Birth	6 mos.	General, many	Indigestion or hard labor	Reflex convulsions or "epilepsy"	Normal	12½ yrs.	12½ yrs.	No more convulsions
51	6 yrs.	18 mos.	4½ yrs.	General, many; petit mal, very many	Constipation	"Reflex convulsions" or epilepsy	Forceps	12½ yrs.	12½ yrs.	No more convulsions
52	16 mos.	15 mos.	3 wks.	General, 2	Indigestion	Reflex convulsions	Normal	14 yrs.	14 yrs.	No more attacks, "cured by dilatation of anus"
53	7¾ yrs.	4 yrs.	3¾ yrs.	Petit mal, frequent	None	Epilepsy	?	14 yrs.	14 yrs.	No more convulsions
54	10 wks.	8 wks.	2 wks.	General, 25 to 100 a day	Indigestion	"Reflex convulsions" or congenital cerebral lesion	Normal Normal	15 yrs. 15½ yrs.	14 yrs. 15½ yrs.	Persisted for 1 year No more convulsions
55	2 mos.	3 wks.	5 wks.	General, 2	Indigestion	Reflex convulsions	?	18 yrs.	18 yrs.	No more convulsions

can be drawn at the time from the severity or the duration of the convulsions as to whether epilepsy will develop later or not.

3. *Cases in Which There was a Single Convulsion or a Series of Convulsions at the Onset of Some Acute Disease or With an Attack of Acute Indigestion.*—There were thirty-nine cases in this class. The severity of the convulsions varied materially. In some cases there was only one slight convulsion, while in others the children were in a convulsive state for from two to four days. There were all degrees of severity between these two extremes. Thirty of these patients were infants, six were between 2 and 5 years of age, and three were between 5 and 10 years of age.

Two of these children are now epileptic and feebleminded. One of them was in a convulsive state for thirty-six hours as the result of indigestion. The other one, a premature infant, had a series of slight convulsions when four days old without apparent cause. A third child has petit mal. This child had two sets of convulsions two weeks apart, each lasting less than twelve hours. Both sets were attributed to indigestion, which he undoubtedly had at the time. Another baby that had its convulsions when six months of age continued to have "staring spells" for six months, which were called petit mal by some physicians and reflex from gas by others. This child had a convulsion with the onset of mumps when 2½ years of age, but has had none in the last year. It hardly seems fair to call him well after so short a period. Thirty-five patients have not had any more convulsions and are now apparently well. The time elapsed since the convulsions in these cases has been two, two and one-half, two and one-half, two and one-half, three, three and one-half, four and one-half, four and one-half, five, five and one-half, six, six and onehalf, seven and one-half, eight, nine and one-half, ten, ten and one-half, ten and one-half, eleven, eleven and one-half, eleven and one-half, eleven and one-half, twelve, twelve and one-half, twelve and one-half, thirteen, thirteen and one-half, fourteen, fourteen and one-half, fourteen and one-half, fourteen and one-half, fifteen, eighteen, eighteen and one-half and nineteen years, respectively. It may be said that it is not safe to consider these children well unless at least five years have elapsed since the convulsions. On this basis twenty-seven can be regarded as absolutely well.

4. *Cases in Which There Had Been Repeated Convulsions During a Considerable Period or in Which There Had Been Repeated Attacks Suggesting Petit Mal.*—There were fifty-five cases in this class. Seventeen of these children are now epileptics, and five of the seventeen are feebleminded. Three have only attacks of petit mal. The others have

grand mal. The children who now have petit mal, all had grand mal when they were seen, and the children who had petit mal when they were first seen, now all have grand mal. In one instance grand mal developed after a free interval of seven years, while in another case petit mal developed after a free interval of six years. Thirty-eight patients are now well. Twenty-two patients had no more attacks. The time elapsed in these cases since they were seen has been three, three and one half, four, five and one-half, seven, seven and one-half, nine, nine, nine, nine, nine and one-half, ten, eleven and one-half, eleven and one-half, twelve, twelve, twelve and one-half, twelve and one-half, fourteen, fourteen, fifteen and one-half and eighteen years, respectively. The intervals since the last convulsions in the other sixteen cases are two, two, three and one-half, four and one-half, four and one-half, four and one-half, five, five and one-half, five and one-half, five and one-half, six, seven, eight and one-half, eleven and one-half, twelve and fourteen years, respectively. As one case developed grand mal after an interval of seven years, it may, perhaps, be said that it is not safe to call a child well until seven years have elapsed. If seven years is taken as an arbitrary standard, only twenty-three of these patients can be said to be absolutely safe.

I have endeavored by analyzing these cases to determine if there was anything in the history or character of the convulsions in these cases which would aid in determining in a given case whether epilepsy was likely to result in the future or not. The results of this analysis have, however, been rather unsatisfactory. There was (as is shown by Table 2) a somewhat larger proportion of hard labors or deliveries by forceps in the cases which developed epilepsy than in those which did not.

TABLE 2.—CONDITIONS AT BIRTH

Labor	Epileptics per Cent.	Not Epileptics, per Cent.
Normal	47	53
Hard or forceps	41	32
Cesarean	..	2
Unknown	12	13

Table 3 shows the character of the attacks and the relative proportions of mild and severe forms in round numbers in the two classes of cases.

TABLE 3.—CHARACTER OF ATTACKS

Character of Attacks	Epileptics per Cent.	Not Epileptics, per Cent.
General convulsions	65	71
Petit mal	30	16
Both	6	13

There was, therefore, a somewhat larger proportion of general convulsions in those that did not develop epilepsy, while nearly two-thirds of the children that had attacks classified as "petit mal" developed epilepsy. That is to say, these figures seem to show that epilepsy is more likely to develop in children who have attacks classified as "petit mal" than in those who have general convulsions. This conclusion hardly seems a reasonable one. The conclusion is justified, however, that repeated attacks, which would be classified as petit mal, are as likely to be followed by epilepsy as are repeated attacks of general convulsions. The age of the patients at the onset of the attacks was below two years in 70 per cent. of both classes, while convulsions at birth were twice as numerous in the cases in which epilepsy did not develop later as in those that did. These figures seem to show that babies who have convulsions at birth are not as likely to develop epilepsy as those who first have them later. This conclusion may or may not be justifiable. It is reasonable to conclude from them, however, that convulsions at birth are not as likely to result in epilepsy as has been commonly supposed.

The apparent etiology in these cases at the time when they were seen, as far as there were enough of a kind to make it worth while to group them, is shown in Table 4.

TABLE 4.—APPARENT ETIOLOGY WHEN SEEN

Apparent Etiology	Epileptics per Cent.	Not Epileptics, per Cent.*
Digestive tract	35	82
Injuries or hard labor	23	8
None	30	11

* Several possible causes in a number of cases.

This table seems to show fairly definitely that when there is no apparent etiology, or when the cause of the attacks is apparently an injury or severe labor, epilepsy is far more likely to develop than when the apparent cause is a disturbance in the digestive tract. That is, the absence of any apparent cause or the history of an injury or of a hard labor, in the absence of any other apparent cause, makes the development of epilepsy later quite probable. If the apparent cause is indigestion, the chances are more than two to one that epilepsy will not develop.

Table 5 shows the duration of the attacks before the patients were seen.

TABLE 5.—DURATION OF ATTACKS WHEN SEEN

Duration When Seen	Epileptics per Cent.	Not Epileptics, per Cent.
Less than 1 year	30	66
From 1 to 5 years	40	31
More than 5 years	30	3

It is very evident from this table that the longer the attacks have persisted, the more likely they are to be epilepsy or to eventuate in epilepsy.

While the analysis of these cases shows little which is of apparent value in determining whether or not epilepsy will develop later in children who have had repeated convulsions or repeated attacks classified as petit mal, it seems from an analysis of the diagnoses which were made at the time the children were first seen as if there was something about the impression which was obtained or about the appearance of the children which was of more importance than anything which is shown in the analysis of the various points which have been considered. Table 6 is interesting in this connection.

TABLE 6.—DIAGNOSES WHEN SEEN

Diagnosis When Seen	Epileptics per Cent.	Not Epileptics, per Cent.
Convulsions from fright	6	..
Reflex convulsions	6	53
Reflex convulsions or congenital cerebral	..	3
"Reflex convulsions" or epilepsy	29	24
"Other conditions" or epilepsy	6	5
Reflex convulsions or epilepsy	6	..
"Epilepsy" or reflex convulsions	12	3
"Epilepsy" or other conditions	..	3
Epilepsy and petit mal	35	8

The condition quoted is the more probable diagnosis. Percentages are given in round numbers.

This table shows many mistakes in diagnosis, which is not surprising. It shows also that epilepsy was considered in 88 per cent. of the children that developed epilepsy and in only 43 per cent. of those that did not develop epilepsy, and that epilepsy was the first choice in 47 per cent. of those that became epileptic and in only 14 per cent. of those that did not. The flat diagnosis of reflex convulsions was made in only 6 per cent. of those who became epileptic, while it was made in 53 per cent. of those who did not develop the disease. On the other hand, the flat diagnosis of epilepsy was made in 35 per cent. of those who developed the disease and in only 8 per cent. of those who did not.

COMMENT AND CONCLUSIONS

The results of this study are most unsatisfactory and very few conclusions can be drawn from it. The following conclusions are, perhaps, justifiable to a certain extent. Convulsions which are a manifestation of spasmophilia are not likely to eventuate in epilepsy. Convulsions which occur in the course of whooping cough must always be regarded seriously, as they are quite likely to be followed by epilepsy later. Single convulsions or a series of convulsions occurring at the onset of an acute disease or with an attack of acute indigestion are less

likely to be followed by epilepsy than are repeated convulsions during a considerable period or repeated attacks suggesting petit mal. Repeated attacks which would be classified as petit mal, or which suggest it, are just as likely to eventuate in epilepsy as repeated attacks of general convulsions. Nothing can be told from the nature of the early attacks as to the nature of the attacks when epilepsy develops later. When an injury to the head has directly preceded the onset of the attacks or there is no apparent cause for the attacks, epilepsy is more probable than when there is an apparent cause, such as indigestion, for each attack. The presence of an apparent cause for the attacks does not, however, exclude epilepsy. The longer the attacks have persisted, the more probable is the diagnosis of epilepsy. General impressions, which cannot be explained, have a certain value in diagnosis. Finally, and most positively, there is no way to determine immediately when a baby or child has a convulsion, or has had repeated convulsions or repeated attacks suggesting petit mal, whether it has epilepsy or whether it will develop it later.

POSTINFLUENZAL ENCEPHALITIS *

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INTRODUCTION

The pandemic of influenza which in the fall of last year spread with such alarming virulence over two continents spared no age. Though perhaps less frightful in its ravages among infants and children, there were many serious complications. Among these was a group of very unusual symptoms related to the central nervous system. Sudden onset, two or three weeks after an attack of influenza, of stupor or coma, paralysis with frequent cranial nerve involvement and a normal cerebrospinal fluid were the most significant manifestations. A study of eight such cases from the Pediatric Service of Mount Sinai Hospital form the basis of this paper.

HISTORY

To modern observers, it seemed for a while to be a new symptom complex, but as early as 1712, during an epidemic of influenza, Zuelzer[1] made note of peculiar drowsy states complicating many cases. In the course of the extensive epidemic of 1890 numerous references were made to similar nervous conditions. Henoch[2] described a case of polioencephalitis with paralysis of one arm which developed in a girl, 11 years of age, two weeks after an attack of influenza. Remak[3] noted a fatal case of bulbar paralysis in a child immediately after influenza. A case of stupor, convergent strabismus and hemiplegia following influenza in a child, 3½ years of age, was described by Knotten.[4] The same author notes a case of polioencephalitis in a child, 4½ years of age, during the epidemic. Alfred Brionne[5] refers to a case of polioencephalitis with paralysis of deglutition, reported by Uhthof. Otto Leichtenstern,[6] in 1896, was the first to give a complete

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1. Zuelzer: Influenza, Ziemssen's Handb. d. Speziellen Pathol. und Therap. **2**:506, Part 2, 1874.
2. Henoch: Berl. klin. Wchnschr., No. 8, 1890.
3. Remak: Quoted by Bosseks; Die Geschichte der Influenza und ihre Nervöse u. Psychische Wachkrankheiten. Leiden, E. Ijdo, 1894.
4. Knotten: Quoted by Kohts; Ueber Paralyse u. Pseudo-Paralyse im Kindesalter nach Influenza.
5. Brionne, Alfred: Contribution a l'étude de la forme nerveuse de la grippe et de ses complications. Paris, H. Jouve, 1890, 62, p. 14, No. 378.
6. Leichtenstern, Otto: Influenza and Dengue, Wien., Nothnagel **4**:118, 1896.

detailed account of the condition. Following the recent epidemic a large number of cases were observed in England and France. An exhaustive report was presented before the Royal Society of Medicine last year. F. G. Crookshank[7] gave statistics of 127 cases that came under his observation in the London and North West London Hospitals. Fifty-seven of these cases occurred in persons under 10 years of age. Drowsiness was noted in thirty, stupor or prolonged lethargy in twenty-six, "unconsciousness" or coma in twenty-five cases. Paresis or paralyses of limbs were present in forty-seven cases and ptosis was marked in twenty-three cases. Neal[8] described cases occurring among children in New York.

TERMINOLOGY

The first term applied to this syndrome was "sleeping sickness" as described in the epidemic of 1712 in Tübingen by Zuelzer. Such a nomenclature would confound the condition with trypanosomiasis. During the epidemic of 1890 reference was frequently made to nona, a meaningless term, which was soon forgotten. "Grippal encephalitis" was used by Leichtenstern. "Lethargic encephalitis" was suggested by Economo[9] in 1917, but this has been criticized by Bassoe[10] as being illogical as the patient not the encephalitis is lethargic. He proposes the term "epidemic encephalitis." We believe from a study of the relation of this condition to influenza that the most appropriate designation is postinfluenzal encephalitis.

ETIOLOGY

One cannot fail to be struck by the close association of this symptom complex with epidemics of influenza. The only periods during which it has been observed have been the years following the extensive epidemics. Pfuhl[11] has been able to isolate the influenza bacillus from the brain of one of these patients. This finding, however, has not been confirmed. All our own cases were preceded by attacks of influenza.

PATHOLOGY

Leichtenstern, in 1896, described the condition as a hemorrhagic encephalitis. A most careful and detailed account of the pathologic changes has been contributed by M. Marinesco[12] at Bucharest. He examined two specimens. Grossly disseminated punctiform hemor-

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7. Crookshank, F. G.: Brit. M. J. **2**:489 (Nov. 2) 1918.
8. Neal, Josephine B.: Meningeal Conditions Noted During the Epidemic of Influenza, J. A. M. A. **72**:714 (March 8) 1919.
9. Economo: Wien. klin. Wchnschr., July 26, 1917.
10. Bassoe: J. A. M. A. **72**:972 (April 5) 1919.
11. Pfuhl: Berl. klin. Wchnschr., p. 979, 1892.
12. Quoted by Lieut.-Col. F. W. Mott, Brit. M. J. **2**:489 (Nov. 2) 1918.

rhages in the gray matter about the floor of the fourth ventricle, the acqueduct of Sylvius and in the posterior part of the pons and the peduncles were found. The cerebral cortex showed only congestion of some of the vessels of the leptomeninges. Microscopically, he found four kinds of lesions: 1. Infiltration of the walls of the small vessels, especially the veins with lymphocytes and plasma cells in the adventitia in several layers. 2. Foci of interstitial inflammation formed of neuroglia cells and large cells with eccentric nuclei, lymphocytes and polynuclears. 3. In some of the nerve cells there was dissolution of Nissl's bodies, relative achromatosis, reduction in volume of cell body and of the number of prolongations. 4. Numerous foci of hemorrhages around the walls of the small blood vessels. We were unable to make any postmortem pathologic examinations as none of our patients succumbed to the disease.

SYMPTOMATOLOGY

From our own observations of the cases we were able to differentiate three main forms, depending on the severity and the most prominent symptoms. We may group them into the (1) irritable, (2) lethargic and (3) lethargic with paralyses. The irritable type is characterized by marked restlessness, excitability and almost continuous crying.

A girl, three years of age, was admitted to the hospital with a history of influenza two weeks previously. Four days before admission the mother noticed the onset of spells lasting about one-half hour and appearing every three or four hours during which the child would cry and shout in wild excitement. This continued for several days in the ward. There were no convulsions and no paralyses. The temperature ranged between 97.8 and 101 F. The blood count showed a moderate leukocytosis, with 13,800 leukocytes; polymorphonuclears, 74 per cent.; lymphocytes, 25 per cent.; transitionals, 1 per cent. The cerebrospinal fluid was normal. The patient recovered completely in ten days. This represents the mildest form.

In the lethargic form the most prominent feature, as the name implies, is a drowsy state. The facial features are expressionless, masklike, resembling the Parkinson syndrome. The patient lies in a semicomatose condition for weeks, resents being aroused and takes nourishment with great difficulty. There may be convulsions or twitchings of the face muscles. There is frequently moderate rigidity of the neck. Most of our cases fell into the third group of lethargy with paralyses. In this type we find, in addition to the stuporous state, some form of paralysis and frequently convulsions. There may be an involvement of the extremities or cranial nerve palsies.

Age.—The youngest patient observed by us was four months of age, the oldest thirteen and a half years; three patient were under one year of age; four were under three years.

Sex.—In our series the cases were equally divided between the two sexes. Crookshank's cases included thirty-three males and twenty-four females.

Mode of Onset.—In all our cases there was a history of previous influenzal infection. In one child the nervous syndrome followed immediately, and in another four months had elapsed. The average interval was about two weeks. In six of the cases the onset of the encephalitis was sudden, in two there was a gradual progressive development. Convulsions were the first manifestations in three cases, stupor in four, marked irritability in one.

Physical.—In half the cases there was some degree of rigidity of the neck. Facial paralysis was present in three, difficulty in deglutition in two cases. In four cases there was weakness of the neck and back muscles, making it impossible to hold the head erect or to sit up. There was a spasticity of the lower extremities with increased knee jerks in three, twitching in one case. One case presented a bilateral internal strabismus, another marked nystagmus. Drowsiness or stupor continued as a prominent symptom throughout the course of the illness in four cases, irritability in three. The masklike expression was present in a more or less degree in the majority of our cases.

Temperature.—Fever has played but a small rôle in all of the cases reported. Rarely has there been a rise of more than two or three degrees above normal. In one of our cases it was as high as 102 F., in another 101 F. The remainder ranged below 100 F. The pulse and respirations were not unusually high.

Fundi.—The fundus of the eye was examined in all of our cases, but showed nothing characteristic. In a few instances there was slight pallor of the disks. In one case optic atrophy developed late in the disease.

Cerebrospinal Fluid.—Of the eight specimens examined, only one showed definite changes. There were eighty cells per c.mm., 80 per cent. of these being lymphocytes. Albumin was present, forming a 1 mm. ring. Fehling's reaction was present. No organisms were found. One other case showed a somewhat increased pressure. The remaining specimens were all within normal limits. In a case reported by Ely,[13] anaerobic as well as aerobic cultures of the fluid were negative. Among Crookshank's cases over 80 per cent. showed some increase in the cell count.

13. Ely: J. A. M. A. **72**:985 (April 5) 1919.

Blood Counts.—Marked leukocytosis was present, in one case being as high as 34,000 cells, with a differential of 84 per cent. polymorphonuclears, 12 per cent. small lymphocytes and 4 per cent. large mononuclears. In two cases a normal blood picture was present, and the remainder showed a moderate leukocytosis ranging between 9,000 and 13,800.

PROGNOSIS

When the cases first appeared with the marked lethargy and frequent paralyses, the prognosis suggested was usually bad. But we have found it much better than the alarming state of the patient seemed to indicate. Thus far, we have not lost any cases. In at least two cases, however, the mentality has been definitely affected. The children have become imbecilic. In this respect the condition differs from the polioencephalitic form of infantile paralysis in which the mentality is rarely ever markedly changed. In three others the paralyses, though considerably improved, have not yet disappeared completely. The irritable type of case recovers rapidly.

TREATMENT

There is no specific therapy. Our treatment has been symptomatic throughout. For the irritable type mixed bromids and chloral are of some value. In the paralytic type massage of the involved muscles is of distinct advantage. Good results have been reported from lumbar punctures.[13] We have not found this procedure of special benefit in our therapy.

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THE CONSTIPATING QUALITIES OF ORANGE JUICE*

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Orange juice has long been known in medical textbooks and journals as an efficient and marked antiscorbutic. In some textbooks and in many works for nurses and mothers it has also been given recognition with other fruit juices as being a laxative or mild cathartic. But aside from these general statements the literature is barren of definite data giving an idea of its true position as a laxative or mild cathartic.

Recently McCollum and Pitz[1] have claimed that the antiscorbutic effect of orange juice was due to its cathartic properties. One of us soon afterward doubted the validity of this argument and at the same time first called attention[2] to the presence of oliguria in some cases of scurvy and to the marked diuretic effect of orange juice in some of these. It was believed, however, that this very marked diuretic effect of orange juice in the oliguric cases of scurvy was not due principally to the presence of citric acid or potassium citrate in the orange juice as Hess[3] later claimed, but to a specific therapeutic change in the mineral metabolism or renal function instituted by the definite antiscorbutic effect of orange juice, an effect which potassium citrate in large doses was unable to bring about.

In normal nonscorbutic individuals one would undoubtedly be justified in giving the entire credit for any diuresis produced by the giving of orange juice to its potassium citrate and citric acid contents. From our own experience we believe that orange juice practically never has a cathartic effect, rarely has a laxative effect, often has no effect, and frequently has a constipating effect. It is to this not uncommon constipating effect of orange juice that we wish to call attention by this article.

This nonlaxative and especially constipating effect of orange juice is, we believe, a secondary result of the diuretic action, that is, as more

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* From the Babies' Dispensary and Hospital, the Departments of Pediatrics of Western Reserve Medical School and of Lakeside Hospital.

1. McCollum, E. V., and Pitz, W.: The "Vitamin" Hypothesis and Deficiency Diseases. A Study of Experimental Scurvy, J. Biol. Chem. **31**:229 (July) 1917.

2. Gerstenberger, H. J.: Pathogenesis of Infantile Scurvy; an Hypothesis, Trans. Am. Pediat. Soc. **29**:187, 1917; Am. J. Med. Sc. **155**:253 (Feb.) 1918.

3. Hess, Alfred F.: Infantile Scurvy; V. A Study of Its Pathogenesis, Am. J. Dis. Child. **14**:337 (Nov.) 1917.

water is passing out through the kidneys less is passed out through the intestines. We have also had the same experience of producing constipation in cases of pyelitis and pyelocystitis which were treated with large doses of potassium citrate. During the summer of 1918 we gave anywhere from 10 to 60 c.c., but usually 20 to 30 c.c. of orange juice daily to infants ill with chronic and acute nutritional disturbances, the latter mainly of the dyspepsia type, without being able to detect clinically any distinct laxative and especially not a cathartic effect. We gave orange juice in these cases because we felt that it might be of value in removing a possible clinically not yet recognizable scurvy in these artificially fed infants, and because of our experience with the giving of orange juice to babies who were constipated while fed with S. M. A. In these latter infants the constipation was usually increased and only rarely seemingly improved by an increase in the dose of orange juice. We have also given in 1 c.c. to 10 c.c. doses of orange juice alone or, when too sour, together with an added amount of sodium bicarbonate, to premature infants weighing as little as 1,500 gm., without producing a laxative or cathartic effect.

Our practical experience with orange juice has been contrary to the generally accepted view that orange juice is a laxative or a cathartic. And as it is given even today by mothers and many physicians for its laxative effect rather than for its antiscorbutic effect, and as we feel that it should practically never be given as a laxative, but only as an antiscorbutic or, in special cases like pyelocystitis, as a diuretic, we have made careful record of the fluid intake of a contented, happy, healthy boy of ten months, and his percentage output by way of the intestines, on the one hand, and by way of the kidneys on the other hand. The amount of water lost by the skin and lungs was not taken into consideration, but inasmuch as the child remained well during the entire period and was constantly happy and contented and of a rather uniform, generally good, behavior and activity we feel that in the case of this child, at least, the loss of water by the skin and lungs would not be a factor that would materially modify our findings.

This infant has been in the children's ward of Lakeside Hospital since he was eight weeks of age, and has continually been fed with S. M. A., on which he has thrived very well. So he was continued on the same food during this observation. In order to avoid any variations in the salt and sugar content of the food, a large batch of S. M. A. was prepared at one time, sterilized in bottles under pressure and refilled to the original amount by the addition of distilled water, which was sterilized in the same autoclave and at the same time with the milk. The bottles were then sealed aseptically, cooled, placed in an icebox at about 5 C., and turned upside down daily. Of this food he

was offered 1,000 c.c. daily, and in addition 100 c.c. of distilled water. His intake of these liquids was practically the same for the comparative period, as can be seen from the accompanying table which gives in detail a summary of the conditions and the results of the experiment. The only definite change made in the diet was the substitution, at the

ANALYSIS OF FLUID INTAKE AND OUTPUT BY INTESTINES AND KIDNEYS

Period	1a	1b	2a	2b	3a	3b
Date of each period	2/1 to 2/5	2/10 to 2/14	2/20 to 2/25	2/27 to 3/4	2/22 to 2/25	3/1 to 3/4
Number of days in each period	4	4	5	5	3	3
Type of experimental fluid	Orange juice	Sugar solution	Sugar solution	Orange juice	Sugar solution	Orange juice
Average daily intake of experimental fluid	C.c. 60.0	C.c. 63.0	C.c. 89.6	C.c. 87.0	C.c. 89.3	C.c. 90.0
Average daily intake of milk and water	1114.0	1145.7	1111.9	1041.5	1112.0	1054.0
Average daily intake of fluid	1174.1	1208.7	1201.5	1128.5	1201.3	1144.0
Average daily weight of moist feces	25.15	26.1	32.92	25.28	44.9	29.16
Average daily weight of dried feces	6.37	6.70	7.28	5.78	10.06	7.80
Average daily amount of water in feces	18.77	19.42	25.88	19.50	34.80	21.33
Average daily output of the kidneys	693.75	719.00	720.60	691.00	776.60	726.30
Total daily output of water by kidneys and stool	712.52	738.42	746.48	710.50	811.40	747.63
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.
$\frac{\text{Aver. daily intake of fluid}}{100} = \frac{\text{Aver. daily urine}}{x}$	59.08	59.48	59.97	61.22	64.64	63.48
$\frac{\text{Aver. daily intake}}{100} = \frac{\text{Av. daily amt. of water in feces}}{x}$	1.59	1.60	2.15	1.72	2.89	1.86
$\frac{\text{Aver. daily intake}}{100} = \frac{\text{Total daily output in urine and stool}}{x}$	60.61	61.09	62.13	62.96	67.54	65.35
$\frac{\text{Total daily output urine and stool}}{100} = \frac{\text{Aver. daily urine}}{x}$	97.36	97.37	96.53	97.25	95.71	97.15
$\frac{\text{Total daily output urine and stool}}{100} = \frac{\text{Aver. daily fecal water}}{x}$	2.63	2.63	3.47	2.74	4.29	2.85

The authors are indebted to F. J. Gallagher, M.D. and to B. E. Levion, A.B. for help in collecting the above data.

proper time, of a 10 per cent. sugar solution composed of 6.5 per cent. dextrose (levulose was not obtainable) and 3.5 per cent. sucrose, for an equal amount of orange juice which had at least the same amount of sugar and in addition the regular salts of orange juice. The stools were collected and weighed in their normal state, then dried at first with a fan, and later to constant weight in an oven with a temperature of 98 to 100 C. Before specimens were selected to determine the effect

of the change in the diet at least twenty-four hours were allowed to elapse in order to avoid overlapping of influences of the liquids.

In the first observation there was no difference ascertainable between the effect of the two solutions; in other words, 60 c.c. of orange juice was no more laxative than 60 c.c. of a 10 per cent. sugar solution, equal to a little more than an even teaspoonful of cane sugar distributed evenly over the entire twenty-four hours in six divided doses. In the second experiment, of which a three days' and a five days' period are reported, there is a decided difference between the effect of the two solutions, the orange juice causing a greater percentage of output by way of the kidneys and a smaller percentage by way of the intestines than the sugar solution. The dosage employed in this experiment was larger than in the former and distinctly larger than the amount of orange juice usually employed in practice, which, on an average, is about 30 c.c. for twenty-four hours, so that a cathartic or at least a laxative effect should have been obtained, and one that would have been greater or at least as great as the laxative effect of 90 c.c. of a 10 per cent. sugar solution composed of two parts glucose and one part sucrose. Even 9 gm. of sugar contained in such a 10 per cent. solution is hardly to be considered as a factor in the production of catharsis or a laxative condition when one remembers that the amount has been evenly distributed throughout the twenty-four hours in six doses of 15 c.c. each, and when it has been given to a normal, healthy boy of ten months of age.

CONCLUSION

Observations made on a normal infant of ten months of age to ascertain the relative position as a cathartic or laxative of orange juice to an equal amount of a 10 per cent. sugar solution composed of 6.5 per cent. glucose and 3.5 per cent. sucrose show in one period no difference between the two solutions, and in the other period a relative laxative advantage of the sugar solution over the orange juice, or better, a relative constipating ability of orange juice as compared with the effect obtained with the 10 per cent. sugar solution. During the sugar solution period anywhere from 95.71 to 96.53 per cent. of the water output went by way of the kidneys, and from 4.29 to 3.47 per cent. went through the intestines, while in the case of the orange juice from 97.15 to 97.25 per cent. of the fluid output went by way of the kidneys, and from 2.85 to 2.74 per cent. by way of the intestines. In other words, orange juice, relatively speaking, has been less laxative than a 10 per cent. sugar solution when given in doses of 15 c.c., six times in twenty-four hours.

This observation confirms our practical experience that orange juice in the maximum amounts ordinarily used has more of a con-

stipating than a laxative effect, and, therefore, should only be used as an antiscorbutic or as a diuretic, but not as a laxative, and especially not for children who are already constipated.

These observations also point to the important rôle that diuresis may play in the production of constipation, and it may be possible that some of the cases of constipation in infants supposed to be due to an abnormally long retention of the feces in the gut and a consequent too complete absorption of water in the large intestine are primarily due to the presence of a factor that causes an abnormal increase in the excretion of water through the kidneys.

A STUDY OF THE LACTOSE, FAT AND PROTEIN CONTENT OF WOMEN'S MILK*

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While the limits and variations in the fat and protein content of human milk are well established, considerable uncertainty still exists regarding the question of the amount of lactose in this fluid,[1] a state of affairs largely due to the fact that while reliable and relatively simple methods for the determination of fat and protein have been in use for many years, the question of the technic of sugar determinations in milk is still unsettled. On account of the relatively large quantities of material required when lactose in milk is to be determined either by the polariscope or by one of the copper reduction methods, it is a more or less general custom, when the amount of material is limited, to calculate lactose by difference after having first determined the total solids, ash, protein and fat, a procedure which throws all the errors of the analysis on the sugar.

Last year Folin and Denis[2] described two methods for the determination of lactose in milk, one a colorimetric procedure based on the reduction of picric acid when treated with sodium carbonate and lactose at the temperature of boiling water, the other a titration method based on the reduction of copper in phosphate solution as described by Folin and McEllroy.[3] These methods have the advantage over most of the procedures now in use, first that by means of them it is possible to obtain results of a considerable degree of accuracy in a relatively short time, and second that only 1 or 2 c.c. of human milk is needed for duplicate determinations.

During the past year we have been engaged by the help of the titration method in the collection of data regarding the lactose content of human milk, and for the sake of completeness have included determinations of the fat and protein.

When the samples of milk were sufficiently large, fat was determined by means of the Babcock method; when the amount of material was limited the analyses were made by Bloor's[4] nephelometric method which requires only 1 c.c. of milk for duplicate determinations.

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* From the Bio-chemical Laboratories and the Department of Pediatrics of the Harvard Medical Schools and of the Massachusetts General Hospital.

1. Holt, Courtney and Fales: Am. J. Dis. Child. **10**:229 (Oct.) 1915.
2. Folin & Denis: J. Biol. Chem. **33**:521 (March) 1918.
3. Folin & McEllroy: loc. cit., 513.
4. Bloor: J. Am. Chem. Soc. **36**:1301, 1914.

Protein was calculated from the total nitrogen values obtained by means of the Kjeldahl-Gunning method, the factor 6.25 being employed.

The experimental work of these investigations was divided into four lines and consisted, first, of a study of the composition of milk at different stages of lactation, the results of which are presented in Table 1; second, of a study of the composition of milk at the beginning and at the end of a single nursing, the results of which are presented in Table 2; third, of a study of the composition of milk taken practically simultaneously from the right and left breasts, the results of which are presented in Table 3, and fourth, of a study of the variations in composition of milk taken at three-hour periods during the course of a single day.

The samples of milk were obtained from the Wet-Nurse Directory, the Boston Eye and Ear Infirmary, the Children's Medical Out-Patient Department of the Massachusetts General Hospital, and from private practice. They were either drawn or expressed from a single breast at a time. The following technic was found to be most satisfactory: draw or express one ounce of milk before the infant is allowed to nurse, then allow the baby to nurse the usual length of time, but never longer than twenty minutes; finally draw or express one ounce of milk after the infant has finished. In this manner samples of the "fore milk" and a sample of the "strippings" are obtained. The figures which are reported later show how essential this is and how faulty conclusions may be drawn from the analysis of milk obtained in an improper manner.

Table 1 gives figures averaged from two samples of milk, one taken before nursing and the other immediately after nursing, except in a few instances, noted in the remarks.

It was found that the amount of lactose was low in colostrum, as has already been described by other investigators. In one instance in which the mother had pneumonia, it was necessary to pump her breasts for six days. The total amount of milk diminished until only one ounce could be obtained on the last day. When the baby was five days old the milk contained 5.8 per cent. sugar; when six days old, the milk contained 5.1 per cent. sugar, and when eleven days old, the milk contained 4.55 per cent. sugar (numbers 75, 76, 77). This sequence of events was probably due to the drying up of the milk rather than to the pneumonia, and is suggestive of what may be expected at the time of weaning. Further investigations are necessary to settle this point. The average amount of lactose in sixty samples was 7.19 per cent. There was a general tendency for the concentration of lactose to increase throughout the period of lactation. During the first ten weeks, two thirds of the samples of milk contained less than 7 per cent.

TABLE 1.—Analyses of Breast Milk at Different Stages of Lactation

No.	Lactose per Cent.	Fat per Cent.	Protein per Cent.	Age of Child	Age of Mother Years	Number of Pregnancies	Remarks
172	3.2	....	....	2 days	..	.....	Colostrum
173	7.5	0.8	....	4 days	..	.....	Has never nursed
176	6.7	3.8	2.1	5 days	..	.....	Milk deep yellow color. Changing from colostrum
75	5.8	....	....	5 days	23	First	Mother has pneumonia. Baby not nursing
76	5.1	1.5	3.7	6 days	..	.....	Same as 75
77	4.85	1.81	2.01	11 days	..	.....	Same as 76
22-23	6.6	1.5	1.57	14 days			
151-2	7.05	3.3	1.30	17 days	26		
147	7.46	2.43	1.59	17 days	24	Third	
149	6.97	3.16	1.83	17 days	..	.....	Seven months premature
72	7.18	1.5	1.97	18 days	20	First	
90	6.45	1.66	1.96	21 days	21	First	Sample taken before nursing
16-17	6.40	5.8	1.18	21 days	16	Second	Baby gaining normally
20	6.73	9.9	1.18	21 days	16	Second	Baby gaining normally
36-7	6.8	1.8	1.40	21 days	20	First	
38	6.93	3.8	1.48	21 days	18	First	Fat and protein sample
47	7.10	6.4	1.48	21 days	..	.....	Sample before nursing
56	7.20	9.5	1.44	21 days	..	.....	Sample before nursing
131	7.07	4.20	1.58	25 days	23	First	
129	7.20	4.00	2.25	26 days	25	Second	
154	6.94	1.81	1.50	25 days	20	First	
42	6.88	4.65	....	4 weeks	..	First	Child has eczema of face
142	7.20	2.6	1.27	4 weeks	..	First	Unknown whether before or after nursing or mixed milk
40	6.96	6.0	1.16	4 weeks	16	Second	Baby gaining, satisfied. Digestion good
43	6.68	7.4	1.05	4 weeks	16	Second	Was given 75 grams lactose 3 hours before nursing
45	6.73	6.0	1.35	4 weeks	18	First	Was given 75 grams lactose 3 hours before nursing
58	6.73	9.2	1.42	4 weeks	18	Second	
60	5.49	6.4	2.23	4 weeks	18	First	Fat and protein from a single sample taken before nursing
156	6.80	3.1	1.63	4 weeks	19	First	
162	6.87	2.18	1.35	4 weeks	21	Third	
145	6.90	3.80	1.48	5 weeks	29	Third	
33	7.05	8.8	1.83	5 weeks	..	Second	Mother producing more milk than baby can take and drawing 12-14 ounces
81	7.00	6.3	1.63	5 weeks	18	Second	
83	6.47	7.05	1.80	5 weeks	22	First	Protein from before nursing, sample only
102	8.20	....	1.56	5 weeks	23	Third	
128	7.2	2.2	1.14	5 weeks	25	First	Single sample taken before nursing
91	6.53	6.36	1.46	6 weeks	16	First	
160	6.04	2.25	1.41	6 weeks	35	Third	
88	6.45	4.9	1.74	7 weeks	21	First	
124	7.8	0.8	1.43	8 weeks	..	.....	Single sample taken before nursing
31	7.43	3.4	1.26	9 weeks	..	Second	
24	6.74	....	....	9 weeks	33	First	
28	6.75	5.0	1.57	9 weeks	38	First	Not quite enough milk for baby
93	6.30	9.9	1.67	9 weeks	22	First	
32	6.94	3.8	1.80	11 weeks	..	Second	Baby weaned because of pneumonia. Milk drawn
136	7.70	5.3	1.18	13 weeks	17	First	
79	7.10	1.64	1.37	18 weeks	28	First	Baby has eczema
135	7.30	0.79	1.05	18 weeks	21	First	Single sample before nursing
153	7.20	3.3	1.05	20 weeks	..	Third	Single sample before nursing
108	8.1	3.0	1.18	22 weeks	..	Second	Baby well. Stools regular with soft curd
117	7.80	2.7	1.22	22 weeks	..	.....	Single sample before nursing. Baby has bronchitis and diarrhea
14	6.45	....	....	25 weeks	..	. ....	Mother has diabetes; urine contains 6.95% sugar
158	7.20	2.40	1.22	26 weeks	35	Third	
4	6.98	....	....	28 weeks	30	First	
7	7.36	....	....	28 weeks	20	First	
8	6.06	....	....	28 weeks	26	Third	
220	7.6	4.00	1.16	29 weeks	35	Third	Normal mother and infant
12	6.76	....	....	30 weeks	24	First	
116	8.10	2.2	1.00	30 weeks	..	.....	Single sample before nursing. Baby well
104	8.00	....	1.14	32 weeks	23	First	Fat in sample after nursing, 8.4%. Baby had one normal stool a day
110	7.70	....	1.00	34 weeks	..	Third	One normal stool a day
133	7.30	9.3	1.06	39 weeks	..	.....	Normal baby with normal stools

TABLE 1.—ANALYSES OF BREAST MILK AT DIFFERENT STAGES OF LACTATION—(*Continued*)

No.	Lactose per Cent.	Fat per Cent.	Protein per Cent.	Age of Child	Age of Mother Years	Number of Pregnancies	Remarks
106	8.35	4.13	1.09	40 weeks	..	Third	Baby 17 pounds. General condition fair; two yellow stools a day
114	8.09	5.33	1.22	40 weeks	..	Sixth	Stools normal
118	7.70	3.9	1.20	43 weeks	..	.....	Baby under weight has bronchitis and green stools
120	7.25	4.25	1.15	49 weeks	..	.....	From 2 to 3 well digested soft yellow stools a day

TABLE 2.—ANALYSIS OF MILK AT THE BEGINNING AND AT THE END OF A SINGLE NURSING

No.	Before			After			Age of Child	Age of Mother, Years	Number of Pregnancies
	Lactose, per Cent.	Fat, per Cent.	Protein, per Cent.	Lactose, per Cent.	Fat, per Cent.	Protein, per Cent.			
16, 17	6.09	5.3	1.18	6.72	6.4	1.18	3 weeks	18	Second
20, 21	6.82	8.9	1.18	6.64	10.9	1.22	3 weeks	18	Second
22, 23	6.62	1.5	1.57	6.77	5.2	1.57	2 weeks	18	First
24, 25	7.23	....	....	6.25	....	....	9 weeks	33	First
28, 29	6.86	5.0	1.57	6.64	13.4	1.57	9 weeks	38	First
30a, 30	7.2	0.58	1.40	6.00	7.40				
34, 35	5.45	6.70	1.84	6.64	7.10	1.57	17 days	18	First
36, 37	6.97	1.8	1.40	6.64	....	....	3 weeks	20	First
38, 39	7.04	3.8	1.48	6.82	....	....	3 weeks	18	First
40, 41	6.79	7.1	1.13	7.13	4.9	1.18	4 weeks	16	Second
43, 44	6.94	7.4	1.05	6.43	7.4	1.05	4 weeks	16	Second
45, 46	7.03	4.0	1.65	6.98	8.0	....	4 weeks	18	First
47, 48	7.10	6.4	1.48	4.88	16.0	....	3 weeks	18	First
49a, b	7.18	2.4	1.44	7.62	8.0	1.52	27 weeks		
56, 57	7.20	9.5	1.44	6.57	....	....	3 weeks	18	First
58, 59	6.82	7.4	1.53	6.64	11.0	1.31	4 weeks	18	Second
60, 61	5.16	6.4	2.23	5.82	....	....	4 weeks	18	First
62, 63	5.82	5.6	1.97	5.96	5.2	2.2			
67, 68	6.4	2.4	1.83	6.47	3.8	1.92			
70, 71	7.58	2.5	1.66	7.40	4.4	1.57	26 weeks	26	Second
72, 73	7.28	1.6	1.97	7.08	1.4	1.98	18 days	20	Second
79, 80	7.50	2.06	1.44	6.7	1.22	1.31	18 weeks	28	First
81, 82	7.0	5.60	1.57	7.0	7.10	1.70	5 weeks	18	Second
83, 84	6.55	5.6	1.80	6.4	9.1	....	5 weeks	22	First
88, 89	6.65	3.2	1.79	6.26	6.6	1.70	7 weeks	21	First
91, 92	6.90	4.52	1.35	6.16	8.00	1.57	6 weeks	16	First
93, 94	6.53	6.48	1.70	6.06	13.3	1.63	9 weeks	22	First
95, 96	6.66	2.48	1.26	6.30	5.40	1.31			
97, 98	6.87	2.76	0.96	6.62	....	0.96			
99, 100	8.80	1.5	1.22	8.40	2.9	1.18			
102, 103	8.20	....	1.43	8.10	....	1.69	5 weeks	23	Third
104, 105	8.00	....	1.14	7.90	8.4	1.10	32 weeks	23	First
106, 107	8.50	3.50	1.09	8.20	4.76	0.89	40 weeks	..	Third
108, 109	8.00	3.20	1.18	8.20	2.70	....	22 weeks	..	Second
110, 111	7.80	....	1.00	7.50	1.09	....	34 weeks	..	Third
112, 113	8.00	4.20	1.35	7.70	7.24	1.44			
114, 115	8.09	0.66	....	8.09	10.0	1.22	40 weeks	..	Sixth
118, 119	7.70	4.20	1.09	7.70	3.6	1.31	43 weeks		
120, 121	7.00	1.5	1.22	7.50	8.0	1.09	49 weeks		
129, 130	7.20	2.9	2.30	7.10	5.1	2.20	26 days	25	Second
131, 132	7.10	4.20	1.66	7.00	4.10	1.50	25 days	23	First
133, 134	7.30	5.50	1.06	7.30	13.1	1.05	39 weeks		
138, 139	7.30	2.9	1.13	6.60	5.5	1.30			
145, 146	7.00	3.0	1.44	6.80	4.5	1.53	10 weeks	2[illegible]	Third
147, 148	7.72	1.66	1.61	7.20	3.20	1.57	3 weeks	24	Third
149, 150	7.08	2.64	1.83	6.86	3.68	1.89	3 weeks	..	First
151, 152	7.20	0.10	0.87	6.90	3.30	1.30	2 weeks	26	Third
154, 155	7.08	1.60	1.40	6.80	2.02	1.40	25 days	20	First
156, 157	6.80	2.60	1.57	6.80	3.60	1.70	4 weeks	19	First
158, 159	7.20	2.00	1.22	7.20	2.80	1.22	26 weeks	35	Third
160, 161	6.30	1.70	1.61	5.78	4.00	1.80	2 weeks	24	First
162, 163	6.54	1.90	1.40	7.20	2.40	1.31	4 weeks	21	Third
180, 181	7.50	8.40	1.60	7.20	8.40	1.70			
182, 183	6.90	1.70	1.60	6.70	4.60	1.90			
184, 185	7.50	1.39	1.35	....	3.68	1.22			
222, 223	7.50	....	1.35	7.50	....	1.35			

lactose. After the tenth week there were four times as many samples which contained between 7 and 8 per cent. as contained less than 7 per cent., and many others which contained over 8 per cent. of lactose. The weakest milk after the colostrum period contained 5.49 per cent. of lactose and the richest 8.35 per cent.

The percentage of fat in the milk of different women varied within wide limits, the lowest mixed milk being 1.5 per cent. and the highest 9.9 per cent. The average amount of fat in the mixed samples was 3 per cent., which is the same as that found in 119 Russian women by Skvortzov,[5] and lower than the 4.5 per cent. found by Engel[6] in ten German wet-nurses. If our wet-nurses only are taken, the average of thirty-five samples is 4.18 per cent. of fat. There does not seem to be any relation between the stage of lactation and the amount of fat in the milk. It seems more probable that the percentage of fat depends more on how completely the breasts are drained and how much of the late milk gets into the sample.

The protein is highest, as would be expected, during the colostrum period. There is a general tendency for the amount of protein to diminish as lactation progresses. It is usually over 1.5 per cent. up to the fourth week and after the twelfth week it averages below 1.2 per cent., the lowest being 1 per cent. So far as we were able to determine with incomplete records, there is no relation between the character of the stools and the amount of total protein.

Table 2 shows the variation in the composition of the milk from the beginning to the end of nursing. There is, as a rule, more lactose at the beginning of nursing than at the end; the difference rarely exceeds 1 per cent.

The percentage of fat, on the other hand, is much greater at the end of the nursing than at the beginning. One example of extreme variation was found in samples 114 and 115 which showed, respectively, 0.66 per cent. fat before nursing, and 10 per cent. after nursing. As a rule, the differences before and after nursing are not so great, and there was usually less than 4 per cent. difference. An occasional sample was very rich both at the beginning and at the end of nursing, as, for example, Nos. 93 and 94, in which the fat was, respectively, 6.48 per cent. before nursing, and 13.3 per cent. after nursing. Occasionally, two samples, one taken before and one after nursing, showed very little change, and in the rare case the milk after nursing showed less fat than before nursing.

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5. Skvortzov: Russk. Vrach. **11**:1392; Ref. Chem. Abstr. **7**: No. 18, 1913.
6. Engel: Sommerfield's Handb. d. Milchkunde, Wiesbaden, 1909, p. 810.

The protein showed less difference in percentage than the fat. It rarely varied more than a few tenths between the beginning and end of nursing. Sometimes it was lower after nursing, and sometimes higher.

The above results emphasize the importance of analyses of milk both before and after nursing, and the uselessness of analyses made (as is too frequently the case) on the first ounce of milk drawn.

Table 3 shows the variation in the composition of the milks taken from the right and left breast of the same woman at about the same time. These figures show that although the milks tend to have the same composition from both breasts, they may be quite different as in Cases 122 and 123, and in Case Cases 18 and 19 where one breast was scarred by a breast abscess.

TABLE 3.—ANALYSIS OF MILK TAKEN SIMULTANEOUSLY FROM THE RIGHT AND LEFT BREAST

No.	Right Breast			Left Breast			Remarks
	Lactose, per Cent.	Fat, per Cent.	Protein, per Cent.	Lactose, per Cent.	Fat, per Cent.	Protein, per Cent.	
85, 87	7.50	2.30	1.60	6.65	6.8	1.60	Right breast taken before nursing; left breast entire quantity
65, 66	7.84	0.73	1.05	7.28	0.63	1.05	Both breasts before nursing
122, 123	7.40	12.0	1.43	7.00	3.80	1.84	Entire amount drawn from both breasts separately
126, 127	7.80	0.7	1.07	7.40	1.4	1.17	
143, 144	7.28	3.5	1.79	7.08	2.4	1.70	
166, 164	7.50	1.6	1.26	7.50	1.5	1.22	Samples taken before nursing } Same
167, 165	7.5	5.0	1.44	7.20	10.0	1.44	Samples taken after nursing } woman
168, 170	7.5	0.80	0.80	7.70	0.8	0.80	Samples taken before nursing } Same
169, 171	7.5	2.4	0.90	7.70	1.9	0.80	Samples taken after nursing } woman
174, 175	6.8	3.3	1.40	7.5	1.8	1.40	Whole of milk drawn, but it did not represent completely empty breasts
30b, 30c	7.2	0.58	1.40	7.2	1.9	1.50	Samples before nursing
18, 19	7.6	2.6	1.18	6.8	8.8	1.01	Right breast normal; left had breast abscess and is scarred

There was a tendency for the volume of the milk to diminish as the day went on, the smallest amount being obtained at 3 p. m. or later. There was very little variation in the amount of lactose and protein, and no rule can be devised from these cases. There were striking variations in the amount of fat, especially in Case 1, and there was a tendency for its concentration to be greater at noon or in the afternoon than at other times of day. There was no relation, however, between the percentage of fat found and the amount of milk drawn from the breast.

TABLE 4.—Analysis of Breast Milk Taken at Three Hour Periods During the Course of a Single Day

Time	Lactose, per Cent.	Fat, per Cent.	Protein, per Cent.	Volume of Milk, C.c.	Remarks
Observation 1	....	....	....	...	Mrs. E. L.: Age of mother 22 years; age of infant 4 months; first pregnancy; normal infant; weight 15 pounds 11 ounces
Nov. 27, 1917					
6 a.m.	7.30	4.0	1.18	180	Breakfast: Cornmeal mush, corned beef hash, muffins, milk and cocoa
9 a.m.	7.25	6.4	1.26	180	Lunch, 10:30 a.m.: Cornmeal gruel with milk
12 m.	7.23	12.5	1.31	170	Dinner: Fish, potato, banana salad, ginger bread
3 p.m.	7.12	8.0	1.22	128	Lunch, 3:30 p.m.: Mellin's food and milk
6 p.m.	7.18	5.3	1.22	157	Supper: Scalloped corn, bread and butter, prunes, cocoa
10 p.m.	7.23	3.6	1.18	150	8 p.m.: Cocoa
Observation 2	....	....	....	...	Mrs. B. R. H.: Age of mother 23 years; baby born May 18, 1918; weight 12 pounds 15 ounces; gained 7 ounces in past week
July 11, 1918					
6 a.m.	6.91	4.00	1.35	202	Breakfast: Oatmeal, hash, potatoes, bread, butter and cocoa
9 a.m.	7.08	4.56	1.40	150	10:30 a.m.: Mellin's food with milk
12 m.	7.08	5.32	1.40	120	Dinner: Hamburg steak, rice, carrots, cottage pudding, cocoa
3 p.m.	7.08	6.02	1.44	130	3:30 p.m.: Cocoa
6 p.m.	7.28	3.62	1.44	120	Macaroni, bread and butter, stewed peaches, cocoa
9 p.m.	7.28	2.84	1.49	90	8:30 p.m.: Cocoa
Observation 3	....	....	....	...	Mrs. M. N.: Age of mother 20 years; baby born May 15, 1918; weight 10 pounds 9 ounces; gained 6 ounces in past week
July 11, 1918					
6 a.m.	7.08	4.73	1.26	90	Food same as in Observation 2
9 a.m.	6.91	3.92	1.35	60	
12 m.	6.91	3.36	1.31	150	
3 p.m.	7.08	3.72	1.35	50	
6 p.m.	7.08	4.60	1.49	90	
9 p.m.	7.08	2.80	1.23	75	
Observation 4	....	....	....	...	Mrs. M. H.: Age of mother 20 years; baby born April 1, 1918; weight 10 pounds 12½ ounces; gained 6 ounces in past week
July 11, 1918					
6 a.m.	6.91	2.00	1.22	135	Food same as in Observation 2
12 m.	6.81	3.72	1.18	172	
3 p.m.	6.91	3.68	1.05	75	
6 p.m.	6.91	4.20	1.09	105	
9 p.m.	6.80	2.48	1.05	75	
Observation 5	....	....	....	...	Mrs. S. McG.: Age of mother 23 years; baby born May 2, 1918; weight 8 pounds 12 ounces; gained 4 ounces in past week
July 11, 1918					
6 a.m.	7.08	2.40	1.67	120	Food same as in Observation 2
9 a.m.	7.08	3.00	1.40		
12 m.	7.20	4.00	1.31	90	
3 p.m.	7.08	3.48	1.40	120	
6 p.m.	7.08	3.48	1.26	75	
9 p.m.	7.09	2.40	1.35	84	
Observation 6	....	....	....	...	Mrs. R. M.: Age of mother 23 years; baby born Dec. 1, 1917; weight 15 pounds 3 ounces; gained 5 ounces in past week
July 11, 1918					
6 a.m.	7.18	2.76	1.05	202	Food same as in Observation 2
9 a.m.	7.00	4.56	1.12	158	
12 m.	6.90	4.56	1.00	120	
3 p.m.	7.20	3.80	1.05	90	
6 p.m.	7.20	3.74	1.00	150	
9 p.m.	7.20	2.85	1.18	120	

SUMMARY

1. Newer chemical methods make it possible to quantitate accurately the fat, lactose and protein from small samples of milk.

2. There is a rapid increase of lactose during the first few days when colostrum changes into milk, and a further increase as lactation progresses. The reverse is true of protein which after the first rapid decrease during the change from colostrum into milk tends to further decrease during the course of lactation. After the colostrum period there does not seem to be any relation between the stage of lactation and the amount of fat in the milk.

3. There is usually a higher percentage of lactose at the beginning of a single nursing than at the end. Although this difference may be one or more per cent., it is usually less. It is almost the rule for the percentage of fat to be much higher at the end of nursing than at the beginning. There is very little, if any, difference in the protein.

4. The milks taken simultaneously from both breasts of the same woman tend to have the same composition, but often vary in respect to the percentage of fat.

5. Toward the middle or later afternoon the volume of milk in a woman tends to diminish. The percentage of fat is as a rule higher at mid-day or mid-afternoon than at other times of day.

THE CAUSE OF A "SPORADIC" CASE OF CEREBRO-SPINAL MENINGITIS*

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This case is presented as a contribution to the epidemiology of cerebrospinal meningitis. A 5-months-old breast fed infant living on a farm developed cerebrospinal meningitis. There was no record of any previous cases in the locality. On account of the isolated location of the farm it was possible to ascertain and examine every person who had come to the house for a week previous. One of the visitors was the uncle of the baby, a man, 33 years of age, who enlisted in the army in April, 1917, and went to France with the Fifty-Second Coast Artillery in August, 1917. He returned to this country in January, 1919, and was discharged from Camp Upton, Jan. 27, 1919. He fondled the baby January 28. A few days later the baby was taken ill and I was called in consultation February 3 by Dr. Reid of Coeymans. Examination of the spinal fluid confirmed the diagnosis of meningococcus meningitis.

The returned soldier said that to his knowledge he had never seen or been in contact with or known of a case of meningitis in his regiment, and had never been sick himself. Miss Stewart, of the New York State Department of Health Laboratory, took postnasal cultures from eight persons which were plated at the farm. All were negative except those from the soldier, and the meningococci obtained from his were agglutinated by the fluid obtained from the spinal fluid of the baby and were also proved to be identical by culture. A point of interest is that the organism still persists in this carrier (June 10). He has used irrigations of all kinds and has been under the treatment of a nose and throat specialist, but the meningococci resist all bacterial procedures. His plight is pitiable. The army refused to take him back. The War Risk Insurance Bureau, the U. S. Public Health Service and the Red Cross all claim they have neither funds nor authority to reimburse him. He is in perfect health, but is held in quarantine and not able to support himself or his family.

All of the textbooks on diseases of children which I have been able to consult mention that cerebrospinal meningitis appears sporadically or in epidemics, implying that there are two forms of the disease.

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Kohls[1] states that the disease has a sporadic tendency without being confined to the region of its development. Zappert[2] alludes to the possibility of animal carriers. "If distributed sparsely they, of course, appear to individual observers as sporadic cases and not actually to be connected with an epidemic. Perhaps the germs are carried by domestic animals and also by vermin."

Holt and Howland[3] sum up the opinion of pediatricians, prevailing in 1914, when the sixth edition of their textbook appeared, as follows: "Sporadic cases of meningitis occur after epidemics and quite apart from them without apparent cause, and it is very exceptional that any connection with a previous case can be established."

The question arises as to what is a sporadic case of disease. In the strict interpretation of the word, there is no such thing, but the term is used to denote isolated or untraced cases. Up until a very short time ago two theories were accepted to account for the appearance of these cases. Infection by or through the air from distant points and by fomites. Scientific investigations and public health experience have disproved the theory of aerial infection. By fomites we do not include instances which are by no means uncommon where there is a direct transference of germs from one person to another on such objects as pencils, cups, fingers, etc., for this is really contact infection. Infection by fomites means by infected objects which retain the infection for a long period of time and over great distances and later give rise to the disease. The experimental work on yellow fever in Cuba which was carried on by Reed and Gorgas proved conclusively that infection could not be transmitted by fomites. The results of investigations of other communicable diseases all point to the fallacy of the fomites theory. Cases which apparently have been traced to fomites can always be traced further to a previous case if full facts are obtainable. Lack of appreciation of modes of infection, both by the laity and medical profession, is responsible for failure to obtain full facts.

Charles V. Chapin[4] says that "laboratory investigation shows that fomites infection with spore bearing bacteria is common; that such infection in typhoid fever, tuberculosis, diphtheria and with other resistant organisms doubtless sometimes takes place; that it is possible in chorea and plague, while such infection in gonorrhea, influenza, cerebrospinal meningitis and pneumonia must be practically impossible."

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1. Deutsche Klinik.
2. Pfaundler and Schlossman's Diseases of Children.
3. Holt and Howland.
4. The Sources and Modes of Infection.

It is difficult to make the public and even members of the medical profession realize that persons, not things, are responsible for the transmission of communicable disease. Many instances of alleged fomites infection were doubtless cases of carrier infection. Persons in whom the germs are growing are much more likely to be the agents of infection than are objects on which the germs are dying.

Hill[5] sums up the present views as follows: "The old public health was concerned with the environment, the new is concerned with the individual. The old sought the sources of infectious diseases in the surroundings of man; the new finds them in man himself. The old public health sought these sources in the air, in the water, in the climate and topography of localities, in the temperature of soils at the depth of four and six feet, in the rise and fall of ground water, and failed because it sought them, very painstakingly and exhaustively it is true, in every place and every thing where they were not. The new public health seeks these sources — and finds them — among those infected persons (or animals) whose excreta or other constituents or body contents enter the bodies of other persons." The old public health also failed to find the routes of transmission of communicable diseases.

The widespread epidemic of poliomyelitis in the summer of 1916 gave abundant opportunity for the study of its epidemiology. Flexner and others were able to definitely prove that healthy carriers could and frequently did transmit the disease. The existence of such carriers had been suspected as otherwise the epidemiology was inexplicable. The striking similarity between poliomyelitis and cerebrospinal meningitis in the mode of transmisson, portal of entry and contagiousness has been pointed out by many investigators.

In this connection it is of interest to note that Kerley[6] in 1916 explained the occurrence of poliomyelitis in isolated and remote distances by healthy carriers and came to the conclusion that the disease may be communicated by personal contact and spread by carriers.

Heiman,[7] in 1917, emphasized the fact that poliomyelitis was spread by innocent carriers. Chapin[8] states that observations by different authorities indicate that a very considerable number of persons who come in contact with cases of cerebrospinal meningitis may carry the organisms on their mucous membranes, yet very few of these persons become infected. He states further that cerebrospinal meningitis extends almost entirely through the agency of carriers.

5. The New Public Health.
6. Kerley: Arch. Pediat. **33**:424 (Jan.) 1916.
7. Heiman: Trans. Am. Pediat. Soc., 1917.
8. Chapin: Loc. cit.

A carrier is a person who harbors disease germs in his body and is capable of infecting others directly or indirectly. There are two types of carriers, active and passive. The active carrier is one who has had the disease but continues to harbor the organisms after clinical recovery. The passive carrier is one who has been in contact with a patient but has at no time suffered from the disease. A more accurate and descriptive nomenclature would be to designate the two types as convalescent and contact carriers. It is not essential, as far as the virulency of the organism is concerned, to distinguish between the two. Simon[9] estimates an average of ten carriers to a patient and as carriers beget carriers it is manifestly impossible to discover them all. The passive or contact carriers are a greater source of danger as their movements are unrestricted and they can disseminate disease over an unlimited territory.

With the advance of scientific research our old pet theories go on the scrap heap while new thories are evolved. Now that transmission of disease through the air and by fomites has been disproved, the present explanation is that communicable disease is transmitted only by means of infected persons, unrecognized cases and healthy carriers.

Epidemiologists claim that an outbreak of communicable disease can be checked with prompt and strict isolation of the sick and early and diligent hunt for carriers, but that after the disease has become established and carriers become numerous isolation and quarantine are of little avail. We were fortunate in being able to detect the carrier and to isolate both the patient and the carrier, and no further cases have developed. If, however, this carrier lived in a large city and had a large number of nieces, nephews and admirers, it is not difficult to imagine a wider distribution of cases with a large number of unrecognized carriers. Isolation, quarantine and disinfection of the sick would not have reached or controlled the situation. We lock the barn door after the horse and everything else has been stolen.

As the disposition and treatment of carriers is very largely a public health measure, a questionnaire was sent to the various state departments of health in order to ascertain their procedure regarding carriers of meningitis.

PROCEDURE REGARDING "CARRIERS" OF MENINGITIS IN THE VARIOUS STATES

Alabama	Isolation until swabs negative. Strict isolation until well.
Arkansas	Isolate until two successive negative examinations are secured. We have not had persistent carriers in this state, except in one or two instances where carriers were released by federal authorities from other states. In that instance no supervision whatever is exercised. One case in question was placed under observation with negative developments.

9. Human Infection Carriers.

California .....No account is taken of meningitis carriers.
Colorado .....Owing to the small number of cases of this disease the problem of carriers has not come before us.
Connecticut .....Shall be isolated until examination of the nasal and throat discharges show the absence of the specific diplococcus.
Florida .....None at present.
Georgia .....No answer to this question.
Idaho .....Nothing relative to carriers.
Indiana .....Isolate them, etc.
Kansas .....Any person afflicted with this disease shall be isolated and quarantined for a period of not less than two weeks nor more than three weeks from onset unless temperature has not returned to normal in the meantime.
Kentucky .....Isolated during an epidemic.
New Orleans.....Isolation until three negative reports, when possible.
Maryland .....Outside of Baltimore City carriers of meningococcus are not looked for as a routine nor are quarantine regulations issued by the state.
Massachusetts ...Under the law the local board of health is directed to make such rules and regulations as are deemed necessary for the control of communicable disease, and our function in the matter would be advisory. We are pleased, of course, where it is practicable, to examine for carriers, and when found to advise as to procedure to render the nasopharynx free from meningococcus.
Minnesota .....None.
Missouri .....No statutory authority. At present time our statutes do not give the state board of health any authority concerning communicable diseases.
New Hampshire..Quarantine.
New Jersey.....Isolation of known cases required. No definite regulations concerning "carriers."
New York.....Contacts excluded from schools and gatherings; and if patient is not isolated, others in the household are quarantined. Carriers subject to special rules and regulations of the state department of health. Patients isolated until three successive cultures from the nasopharynx at intervals of not less than five days, are free of meningococci.
Oklahoma .....Isolation of infected persons and carriers until the nasopharynx is free from the infecting organisms, or at the earliest until one week after fever has subsided.
Pennsylvania ....Wherever we have knowledge of a meningitis carrier we keep the patient isolated until bacteriologic results are negative.
Rhode Island.....No action taken.
South Carolina.. Quarantine carriers and actual cases for three weeks.
South Dakota....Every doubtful case must be classed as of epidemic type and cared for accordingly until proven otherwise.
Texas .....No regular line of procedure has been established.
Vermont .....So few cases in the state recently that it is difficult to make any report on it.
Virginia .....Quarantined when found.
West Virginia....Have had no occasion to do anything. Procedure same as in poliomyelitis.

The answers reveal very little uniformity in the isolation of patients and in dealing with carriers. There are no provisions for the detection of healthy carriers and the compulsory bacteriologic examination of contacts. Public health legislation on this subject is imperative if we are going to limit the spread of communicable disease.

New methods in the control and prevention of communicable disease are bound to result from the change in our knowledge of their transmission. The new public health recognizes the importance of contact and the significance of carriers. The old order of quarantine and disinfection is passing away and a new order of strict personal cleanliness and destruction of body secretions and excreta is at hand.

It is our duty as pediatricians to keep abreast of public health progress and to assist in educating our communities in the new order of things.

From the study of this case we feel justified in claiming that it was caused by contact from a healthy passive carrier, that sporadic cases are in reality untraced cases and that the human carrier is responsible for cases appearing either singly or in epidemics.

FAT METABOLISM OF INFANTS AND YOUNG CHILDREN *

III. FAT IN THE STOOLS OF CHILDREN ON A MIXED DIET

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This study of fat metabolism presents observations on children whose diet contained a large proportion of solid food. It gives the findings as to fat percentage and distribution in the stools of a number of children receivng a mixed diet and the fat retention of these children.

The material examined consisted of 134 collections of the feces of sixty-two children from one to ten years of age. Most of the children were between eighteen months and five years of age. The stools were dried and analyzed according to a procedure we have already described.[1] Only four specimens (Nos. 235, 236, 332, 333) were analyzed by the modified Soxhlet method. The remainder were analyzed according to the modification of the Roese Gottlieb method published by us. The values for fat intake in this paper, although carefully estimated, were not usually obtained by analysis and therefore must be considered as being approximate. When the children were not under our direct supervision, exact directions were given for collecting the stools and a record of the food was obtained for twenty-four hours preceding the collection of feces, as well as for the twenty-four or forty-eight hours period of collection. The fat was determined by the Babcock method in samples of the milk consumed by the children, and in the other food the fat was estimated. When practicable, the fat content of the solid part of the food was determined by taking an aliquot part, which was dried, ground and analyzed by the Roese-Gottlieb method.

The larger number of the children whose stools were examined were in normal condition, both as to general health and digestion. Six of the children were rachitic; eleven were suffering from chronic intestinal indigestion, several being examples of intestinal infantilism.

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1. Am. J. Dis. Child. **17**:6 (June) 1919.

The first part of the paper contains the observations made on healthy children whose digestion was nearly or quite normal; the second part contains the observations on the abnormal cases.

Tables 1, 2, 3, 4 and 5 give the fat per cent. and distribution of fat in the stools of the normal children, and Tables 6, 7, 8, 9 and 10 the retention of fat in most of these cases. In a few instances the stools were not collected for an exactly known period, and consequently the values for retention could not be used.

1. FAT PER CENT. AND DISTRIBUTION OF FAT IN THE STOOLS OF NORMAL CHILDREN

The findings on the stools of several children fed on milk alone or milk with bread and cereal are presented in Table 1. All these children had previously taken a more general diet. Four of the stools were

TABLE 1.—FAT PER CENT. AND DISTRIBUTION IN STOOLS OF CHILDREN ON A DIET OF MILK OR MILK WITH BREAD AND CEREAL

No.	Case	Age	Diet	Intake of Fat, Grams Daily	Analyses of Stools			
					Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
						Soap	Free Fatty Acids	Neutral Fat
226	C. A. 2	2 yr. 3 mo.	35 oz. milk, barley gruel	35.0	33.6	79.7	10.9	9.4
228	E. D. 2	2 yr. 6 mo.	40 oz. milk, 6 tbsp. cereal, 4 pc. bread	42.2	27.1	75.1	14.1	10.8
227	E. D. 1	2 yr. 6 mo.	35 oz. milk	35.0	39.5	71.2	21.2	7.6
225	C. A. 1	2 yr. 3 mo.	35 oz. milk	35.0	45.2	69.0	22.8	8.2
230	P. M.	2 yr. 4 mo.	20 oz. milk, 6 tbsp. cereal	21.1	26.3	64.4	30.7	4.9
231	V. G.	1 yr. 4 mo.	26 oz. skimmed milk, 5 tbsp. cereal, 1 pc. bread	15.0	38.8	61.5	26.9	11.6
233	F. W. 8	3 yr. 7 mo.	30 oz. milk, 4 tbsp. cereal	31.0	34.2	58.7	30.4	10.9
232	J. E.	1 yr. 11 mo.	27 oz. milk, 2 tbsp. cereal, 1 tbsp. vegetable, 2 pc. bread	28.0	20.0	57.2	19.1	23.7
229	M. S.	10 yr.	32 oz. milk	32.0	24.7	40.7	37.2	22.1
234	A. L.	3 yr.	20 oz. milk, 6 tbsp. cereal, 2 pc. bread	21.8	17.7	31.0	38.1	30.9
	Average			29.6	30.7	60.9	25.1	14.0

constipated (Nos. 226, 227, 228, 229); the others were of normal consistency.

The average fat per cent. of dried weight, 30.7, shown in Table 1, is lower than that found in the stools of the infants on modifications of cow's milk. The soap per cent. of total fat is, on the whole, high, being highest in three of the stools which were constipated. The average soap per cent. for the group, 60.9, is also lower than that found by us in normal and constipated stools of infants. Fatty acids are higher than neutral fat in every case but one, which was also true in similar stools of the infants. As far as the analyses of their stools are concerned, this group occupies a place between the infants on milk modifications and the older children on a mixed diet.

The great number of the observations reported in this paper were made on the stools of children on a diet consisting of considerable amounts of solid food and usually a pint or more of milk. The findings have been arranged in three groups according to the gross appearance of the stools: those which were constipated (Table 2), those of normal consistency (Table 3) and those not normal (Table 4).

In the food column in these tables we have enumerated the articles which contained any considerable amount of fat, such as milk, butter, eggs, cod liver oil, etc. The remainder of the diet consisted of carbohydrate foods, vegetables and fruit.

TABLE 2.—Fat Per Cent. and Distribution in Stools of Normal Children on a Mixed Diet. Stools Constipated

No.	Case	Age	Diet	Intake of Fat, Grams Daily	Analyses of Stools			
					Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
						Soap	Free Fatty Acids	Neutral Fat
280	B. W. 3	1 yr. 9 mo.	40 oz. milk, egg, C.L.O., etc. ..	55.5	33.7	75.2	11.4	13.4
318	D. L.	1 yr. 2 mo.	25 oz. milk, egg, etc.	33.6	33.6	65.4	23.7	10.9
268	O. W. 2	2 yr. 7 mo.	32 oz. milk, egg, meat, etc. ...	38.5	29.8	64.6	16.5	18.9
308	H. F. 5	1 yr. 1 mo.	24 oz. fat-free milk, egg, butter, etc.	35.2	16.3	61.3	18.8	19.9
326	E. A. 2	7 yr. 5 mo.	19 oz. skimmed milk, meat, butter, C.L.O., etc.	34.0	31.1	56.4	27.8	15.8
320	J. O. 2	3 yr.	32 oz. milk, egg, meat, etc. ...	38.3	17.0	56.2	18.2	25.6
283	B. W. 6	2 yr.	32 oz. milk, egg, etc.	42.1	24.0	55.5	19.5	25.0
305	H. F. 1	1 yr.	32 oz. milk, egg, etc.	34.9	21.5	53.2	22.7	24.1
269	O. W. 5	2 yr. 8 mo.	32 oz. milk, egg, meat, C.L.O. etc.	53.5	21.8	53.0	28.2	18.8
307	H. F. 3	1 yr.	24 oz. skimmed milk, butter, egg, etc.	36.6	18.9	51.2	29.0	19.8
321	F. S.	1 yr. 6 mo.	40 oz. milk, egg, nut butter, meat, etc.	61.0	23.4	51.2	24.1	24.7
270	A. W. 1	4 yr.	32 oz. milk, egg, meat, etc. ...	38.4	13.4	47.1	29.7	23.2
284	B. W. 12	2 yr. 3 mo.	32 oz. milk, egg, etc.	40.5	17.8	46.3	26.9	26.8
306	H. F. 2	1 yr.	26 oz. milk, egg, butter, etc. ..	36.5	19.0	45.5	25.6	28.9
310	R. K. 1	1 yr. 10 mo.	32 oz. milk, egg, etc.	39.3	11.5	41.5	32.3	26.2
324	T. R.	4 yr.	32 oz. milk, egg, meat, etc. ...	38.3	13.5	36.9	32.6	30.5
322	M. J. 1	3 yr.	24 oz. milk, egg, meat, nut butter, etc.	48.2	12.1	30.0	37.5	32.5
290	D. R.	3 yr.	22 oz. milk, nut butter, egg, meat, etc.	44.8	10.5	30.0	17.3	52.7
296	E. H. 1	2 yr. 3 mo.	32 oz. milk, egg, etc.	38.8	16.1	28.9	37.5	33.6
311	R. K. 2	1 yr. 11 mo.	27 oz. milk, nut butter, etc. ...	31.5	16.9	28.9	47.6	23.5
309	H. F. 7	1 yr. 2 mo.	20 oz. milk, butter, egg, etc. ..	39.8	21.0	27.1	29.3	43.6
	Average			40.9	20.1	47.9	26.5	25.6

In Table 2, which reports the findings on the constipated stools, the average for the daily fat intake is 40.9 gm. The fat per cent. of dried weight of the stools averages 20.1, which is much lower than was found when the diet was mainly milk. The average soap per cent. of total fat, 47.9, is also lower than that seen in Table 1, but the soap predominates over fatty acids and neutral fat in all but the last five instances. The average values for neutral fat and fatty acids are nearly the same; sometimes the fatty acids, sometimes the neutral fat shows the larger percentage.

TABLE 3.—Fat Per Cent. and Distribution in Stools of Normal Children on a Mixed Diet. Stools Normal

No.	Case	Age	Diet	Intake of Fat, Grams Daily	Analyses of Stools			
					Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
						Soap	Free Fatty Acids	Neutral Fat
292	H. M.	1 yr. 6 mo.	30 oz. milk, egg, etc.	39.3	27.4	78.6	8.3	13.1
300	F. B. 1	3 yr. 8 mo.	32 oz. milk, egg, meat, etc.	34.3	24.4	70.2	11.9	17.9
282	D. W. 5	1 yr. 11 mo.	30 oz. skimmed milk, egg, C.L.O., etc.	23.3	17.3	67.5	15.4	17.1
281	B. W. 4	1 yr. 10 mo.	40 oz. skimmed milk, egg, C.L.O., etc.	27.1	20.4	63.7	15.3	21.0
267	O. W. 1	2 yr. 6 mo.	32 oz. milk, egg, meat, etc.	37.8	18.0	57.0	22.3	20.7
291	R. K.	2 yr. 1 mo.	27 oz. milk, egg, butter, etc.	42.5	31.2	55.2	19.1	25.7
303	F. B. 4	3 yr. 7 mo.	20 oz. skimmed milk, butter, egg, meat, etc.	41.6	26.6	55.1	23.5	21.4
276	R. L. 2	3 yr.	24 oz. milk, nut butter, egg, meat, etc.	48.1	10.0	54.5	27.2	18.3
353	R. L. 5	4 yr. 2 mo.	16 oz. milk, butter, egg, etc.	40.0	20.7	54.1	22.3	23.6
351	F. B. 5	3 yr. 9 mo.	19.5 oz. skimmed milk, butter, egg, etc.	44.4	20.4	53.5	17.2	29.3
254	E. M. 6	2 yr. 7 mo.	19.5 oz. fat-free milk, butter, egg, etc.	37.7	16.7	52.6	22.6	24.8
345	E. H.	3 yr. 4 mo.	12 oz. milk, butter, meat, etc.	24.5	8.7	52.1	19.9	28.0
314	P. F.	3 yr. 11 mo.	20 oz. bilk, butter, meat, etc.	36.3	13.9	50.8	20.0	29.2
260	D. S. 2	5 yr.	32 oz. milk, egg, meat, C.L.O., etc.	59.0	29.1	50.6	28.7	20.7
352	F. B. 7	3 yr. 9 mo.	19.5 oz. fat-free milk, butter, egg, meat, etc.	41.1	20.7	49.9	21.2	28.9
264	F. W. 9	3 yr. 7 mo.	20 oz. milk, egg, meat, nut butter, etc.	43.6	16.3	49.8	37.1	13.1
275	R. L. 1	3 yr.	24 oz. milk, butter, egg, meat, etc.	48.1	15.4	48.0	32.4	19.6
259	D. S. 1	5 yr.	32 oz. milk, egg, meat, C.L.O., etc.	58.5	22.5	49.2	33.5	17.3
313	E. K.	2 yr.	32 oz. milk, egg, meat, etc.	37.8	17.6	42.2	30.3	27.5
319	J. O. 1	3 yr.	26 oz. milk, nut butter, etc.	30.5	39.1	41.5	39.3	19.2
274	A. W. 5	4 yr. 2 mo.	24 oz. milk, nut butter, egg, meat, etc.	52.1	13.6	41.4	31.6	27.0
289	M. C.	6 yr.	32 oz. milk, eggs, meat, etc.	44.5	16.6	41.4	34.1	24.5
312	R. K. 3	2 yr.	32 oz. milk, egg, meat, etc.	38.3	12.4	41.4	28.5	30.1
287	W. W.	2 yr. 5 mo.	29 oz. milk, butter, egg, meat, etc.	50.9	14.8	41.2	21.6	37.2
330	H. F.	4 yr. 6 mo.	32 oz. milk, egg, meat, etc.	38.3	11.6	41.2	34.2	24.6
354	R. L. 7	4 yr. 3 mo.	19 oz. skimmed milk, butter, egg, etc.	42.3	16.7	41.0	26.4	32.6
271	A. W. 2	4 yr. 1 mo.	24 oz. milk, nut butter, egg, meat, etc.	48.1	17.6	40.9	32.1	27.0
293	E. S.	1 yr. 2 mo.	19 oz. milk, butter, egg, etc.	30.9	18.7	40.7	29.8	29.5
272	A. W. 3	4 yr. 1 mo.	24 oz. milk, egg, corn oil, meat, etc.	52.1	20.6	40.4	33.2	26.4
298	E. M. 3	2 yr. 6 mo.	20 oz. milk, nut butter, egg, meat, etc.	34.1	13.3	37.2	38.3	24.5
273	A. W. 4	4 yr. 1 mo.	24 oz. milk, egg, corn oil, meat, etc.	52.1	20.6	36.2	33.8	30.0
299	E. M. 4	2 yr. 6 mo.	20 oz. skimmed milk, egg, meat, etc.	40.1	17.4	35.6	33.1	31.3
297	E. M. 2	2 yr. 4 mo.	20 oz. milk, butter, egg, meat, etc.	32.6	16.0	35.3	31.7	33.0
295	M. M.	5 yr. 6 mo.	32 oz. milk, egg, meat, etc.	38.8	11.7	34.4	39.2	26.4
302	F. B. 3	3 yr. 9 mo.	24 oz. milk, nut butter, egg, meat, etc.	42.5	21.0	33.6	27.7	38.7
317	K. P.	2 yr. 8 mo.	21 oz. milk, nut butter, etc.	34.5	9.7	33.0	31.5	35.5
355	R. L. 9	4 yr. 3 mo.	19 oz. fat-free milk, butter, egg, etc.	44.6	15.5	32.3	23.5	44.2
288	E. v. S.	5 yr. 7 mo.	17 oz. milk, butter, egg, meat, etc.	49.9	9.6	31.6	31.1	37.3
350	H. J. 2	1 yr. 4 mo.	12 oz. milk, nut butter, meat, etc.	29.1	14.9	30.0	36.5	33.5
325	C. M.	5 yr.	16 oz. milk, butter, egg, meat, etc.	36.8	11.3	24.0	31.8	44.2
348	M. J. 3	3 yr. 4 mo.	14 oz. milk, nut butter, egg, meat, etc.	31.3	19.5	19.4	24.6	56.0
			Average	40.5	18.0	45.1	27.4	27.5

Table 3 includes the analyses of forty-one normal stools of children whose diet contained considerable amounts of milk. In only three instances (Nos. 345, 348, 350) was the daily quantity less than one pint. These stools were all from children in good condition, and the findings may be taken as being fairly representative of what is normal with a general mixed diet. We have regarded as normal a stool which was smooth, homogeneous, not hard, showing no mucous or undigested food. The reaction to litmus of the fresh stools in the groups reported in this table and the two previous ones was in the majority of cases decidedly alkaline; in a few it was amphoteric; in no case was it distinctly acid.

The average fat percentage of dried weight and the distribution of fat of the normal stools, as shown in Table 3, are practically the same as in the constipated stools of children on a similar diet. This corresponds to the results obtained with stools of infants whose diet was milk-modifications, as published in a previous paper. With such infants no difference was shown between normal and constipated stools in the proportions of fat, protein and ash. The chief difference seems to be in the water content. Since with children on a mixed diet, also, the fat percentage of dried weight averaged nearly the same in the constipated and the normal stools, the values for total ash for each kind of stools were averaged. In the constipated stools the average ash was found to be 19.8 per cent. of the dried weight and in the normal stools it was 18.7 per cent. Although the protein percentage was not determined, the similarity between the constipated and normal stools in both fat and ash percentage confirms our belief that the chief difference between the constipated and normal stools, with mixed diet, as with simple milk feeding, is in the water content, and that the chemical composition of the stool has no constant relation to the constipation.

In Table 4 are shown the analyses of a group of stools not normal. All were acid in reaction, some extremely so. All but one was distinctly abnormal in appearance; many showed considerable mucus; almost all contained undigested food and several showed evidences of fermentation. The children nearly all had milk in varying amounts, but in over half the cases the amount of milk consumed was less than one pint daily. However, the average total fat intake was but little less than that seen in Tables 2 and 3.

We have called attention in a previous paper to the fact that in the process of drying the acid stools of nursing infants a change takes place in the relation between the fatty acids and the soaps, probably owing to the volatilization of lower acids, such as acetic and lactic.[2]

2. Am. J. Dis. Child. **17**:241 (April) 1919.

As a result the soaps are higher and the fatty acids lower in the dried stools than in the moist fresh stools, although the amount of total fat is not altered. In the normal alkaline stools of artificially fed children investigation has shown no difference in the total fat or in the distribution of fat between the moist and the dried stool. In the acid stools, however, such as are considered in Table 4, a different distribution of fat is found in the moist stool from that found in the dried

TABLE 4.—FAT PER CENT. AND DISTRIBUTION IN STOOLS OF NORMAL CHILDREN ON A MIXED DIET. STOOLS NOT NORMAL

No.	Case	Age	Diet	Intake of Fat, Grams Daily	Analyses of Stools			
					Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
						Soap	Free Fatty Acids	Neutral Fat
279	B. W. 2	1 yr. 4 mo.	30 oz. milk, egg, C.L.O., etc. ..	41.6	33.4	58.7	28.2	13.1
285	B. W. 13	2 yr. 4 mo.	28 oz. milk, nut butter, egg, etc.	51.9	18.6	50.2	26.1	23.7
301	F. B. 2	3 yr. 9 mo.	24 oz. milk, butter, egg, meat, etc.	43.4	21.6	49.5	20.4	30.1
328	D. D.	1 yr. 8 mo.	24 oz. milk, meat, C.L.O., etc.	41.3	12.0	48.9	35.2	15.9
247	B. M.	4 yr.	6.5 oz. fat-free buttermilk, butter, bacon, etc.	35.6	6.7	45.0	26.3	28.7
286	B. W. 14	2 yr. 4 mo.	28 oz. milk, egg, nut butter, etc.	51.9	20.9	44.6	24.5	30.9
248	E. D.	2 yr. 6 mo.	11.5 oz. fat-free buttermilk, much cereal, vegetable, etc. ..	9.7	20.4	41.4	47.8	10.8
344	K. v. S.	2 yr. 7 mo.	10 oz. milk, butter, egg, meat, etc.	37.7	8.7	33.9	29.1	37.0
346	E. P.	.....	7 oz. milk, butter, meat, egg, etc	29.5	14.7	33.7	36.1	30.2
340	L. S.	3 yr. 2 mo.	10 oz. milk, egg, meat, fish, etc.	30.5	8.2	32.8	27.1	40.1
339	E. A. 1	7 yr.	15 oz. milk, butter, meat, etc.	32.2	11.3	32.5	26.8	40.7
349	H. J. 1	1 yr. 4 mo.	15 oz. milk, nut butter, egg, meat, etc.	35.5	12.9	31.8	37.7	30.5
327	R. M.	4 yr.	32 oz. milk eggs, meat, C.L.O., etc.	50.5	17.1	28.2	44.7	27.1
338	R. N.	5 yr. 10 mo.	No milk; butter, meat, much vegetable, etc.	23.9	4.3	28.1	40.8	31.1
277	R. L. 3	3 yr. 1 mo.	24 oz. milk, nut butter, egg, etc.	52.1	8.4	20.8	40.7	38.5
329	H. D.	5 yr. 10 mo.	32 oz. milk, egg, meat, etc. ...	37.8	6.4	16.5	29.6	53.9
336	R. K.	6 yr.	13 oz. milk, butter, egg, meat, etc.	45.6	14.9	12.9	34.9	52.2
347	M. J. 2	3 yr.	12 oz. milk, nut butter, egg, meat, etc.	32.4	34.3	9.3	8.9	81.8
337	D. K.	4 yr.	13 oz. milk, butter, egg, meat, etc.	46.6	12.2	5.0	29.6	65.4
		Average.....		38.4	15.1	32.8	31.3	35.9

stool. We have not yet determined how constantly and to what extent this change in the relation between fatty acids and soaps takes place in the drying of acid stools or what the factors are which affect the process, but undoubtedly a portion of the free fatty acids is converted into soaps. Hence, the figures in Table 4 show higher soaps and lower fatty acids than would have been found if moist stools had been analyzed.

The fat percentage of dried weight shown in Table 4 averages somewhat lower than in the preceding tables, and there is a larger

number of cases with low values. The neutral fat averages considerably higher than in normal stools, and there are several high values, suggesting that when the stools are of this character there is a lessened degree of fat splitting. The average for fatty acids is also higher than that found in normal stools. When it is considered that this value would be increased if determined in the moist stool, it is evident that in the average for these acid stools fatty acids form the predominating part of the total fat, the soap being the lowest fraction.

It is to be noted that in eleven of the nineteen cases in Table 4 the intake of calcium and other salts was low because of the small quantity of milk taken, the amount of calcium in the other foods being small in comparison with that in milk. The intake of salts, particularly those of calcium, may have some significance in determining the character of the stools, especially affecting the distribution of the fat.

In Table 5 is given a comparison of the averages of Tables 1, 2 and 3 with the results obtained on normal and constipated stools of infants fed on milk modifications, as reported in a previous paper.[1]

TABLE 5.—COMPARISON BETWEEN AVERAGE ANALYSES OF STOOLS OF INFANTS AND OLDER CHILDREN

	Character of Stools	Intake of Fat, Grams Daily	Analyses of Stools			
			Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
				Soap	Free Fatty Acids	Neutral Fat
Infants on usual milk modifications	Normal	24.0	36.2	72.8	16.5	9.4
	Constipated	22.9	36.0	73.8	17.6	9.0
Children on milk or milk with bread or cereal	Normal or constipated	29.6	30.7	60.9	25.1	14.0
Children on mixed diet	Normal	40.5	18.0	45.1	27.4	27.5
	Constipated	40.9	20.1	47.9	26.5	25.6

This table shows strikingly the difference in fat percentage and distribution of fat between the stools when the diet contained a considerable proportion of solid food and those when the diet was mainly milk, both the total fat percentage and the soap percentage being much lower in the stools of children on a mixed diet. Here, also, is shown the close agreement, previously mentioned, in the composition of the normal and the constipated stools when the diet is similar.

In no group studied was there found any constant relation between the amount of the fat intake and either the fat percentage of dried weight or the distribution of fat in the stools.

2. FAT RETENTION OF NORMAL CHILDREN

Table 6 shows the fat excretion and retention of the children taking only milk or milk with bread and cereal. The stools were normal in all but one instance (No. 229), when they were constipated.

The average fat loss shown in Table 6 is 2.76 gm. The percentage of the intake retained averages 90.6, a value corresponding closely to that found for infants receiving milk modifications.

TABLE 6.—FAT RETENTION OF NORMAL CHILDREN ON A DIET OF MILK OR MILK WITH BREAD AND CEREAL. STOOLS NORMAL OR CONSTIPATED. (CF. TABLE 1.)

No.	Case	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake, Retained
225	C. A. 1	35.0	4.10	30.9	88.3
229	M. S.	32.0	1.88	30.1	94.2
233	F. W. 8	31.0	4.10	26.9	86.8
232	J. E.	28.0	1.92	26.1	93.2
234	A. L.	21.8	1.81	20.0	91.8
Average		29.6	2.76	26.8	90.6

TABLE 7.—FAT RETENTION OF NORMAL CHILDREN ON A MIXED DIET; STOOLS CONSTIPATED. (CF. TABLE 2.)

No.	Case	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake, Retained
321	F. S.	61.0	2.46	58.5	96.1
280	B. W. 3	55.5	4.57	50.9	91.7
289	O. W. 5	53.5	3.20	50.3	94.0
322	M. J. 1	48.2	1.04	47.2	97.8
290	D. R.	44.8	1.26	43.5	97.2
283	B. W. 6	42.1	3.36	38.7	92.0
284	B. W. 12	40.5	3.85	36.6	90.5
309	H. F. 7	39.8	2.71	37.1	93.0
296	E. M. 1	38.8	2.29	36.5	94.1
268	O. W. 2	38.5	2.16	36.3	94.4
270	A. W. 1	38.4	1.43	37.0	96.3
320	J. O. 2	38.3	1.20	37.1	96.8
324	T. R.	38.3	1.49	36.8	96.1
307	H. F. 3	36.6	2.55	34.0	92.9
308	H. F. 5	35.2	1.96	33.2	94.3
305	H. F. 1	34.9	1.68	33.2	95.2
326	E. A. 2	34.0	4.17	29.8	87.7
318	D. L.	33.6	4.04	29.6	88.0
311	R. K. 2	31.5	2.36	29.1	92.5
Average		41.2	2.51	38.7	93.9

The fat excretion and the fat retention of children taking a mixed diet are shown in Tables 7, 8 and 9. Table 7 shows the values when the stools were constipated, Table 8 when they were normal, and Table 9 when they were acid abnormal stools.

The average excretion and retention in Tables 7 and 8 are seen to be almost identical. The average fat loss when the stools were constipated is 2.51 per cent.; when they were normal, 2.48 per cent. The percentage of the intake retained is 93.9 for both groups, with only

TABLE 8.—Fat Retention of Normal Children on a Mixed Diet; Stools Normal. (cf. Table 3.)

No.	Case	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake, Retained
260	D. S. 2	59.0	4.71	54.3	92.1
259	D. S. 1	58.5	5.65	52.8	90.4
272	A. W. 3	52.1	2.20	49.9	95.8
273	A. W. 4	52.1	5.07	47.0	90.3
274	A. W. 5	52.1	2.56	49.5	95.2
287	W. W.	50.9	2.40	48.5	95.3
288	E. v. S.	49.9	2.22	47.7	95.7
276	R. L. 2	48.1	1.82	46.3	96.3
271	A. W. 2	48.1	1.21	46.9	97.5
355	R. L. 9	44.6	3.34	41.3	92.6
289	M. C.	44.5	1.70	42.8	96.2
351	F. B. 5	44.4	2.74	41.7	93.8
264	F. W. 9	43.6	2.92	40.7	93.3
354	R. L. 7	43.2	3.29	39.9	92.4
291	R. K.	42.5	2.18	40.3	94.8
302	F. B. 3	42.5	2.25	40.2	94.6
352	F. B. 7	41.1	2.85	38.3	93.2
290	E. M. 4	40.1	2.55	37.6	93.7
353	R. L. 5	40.0	3.57	36.4	91.2
292	H. M.	39.3	2.50	36.8	93.6
295	M. M.	38.8	1.56	37.2	95.9
312	R. K. 3	38.3	0.95	37.3	97.5
330	H. F.	38.3	1.62	36.7	95.8
313	E. K.	37.8	3.57	34.2	90.6
267	O. W. 1	37.8	1.89	35.9	95.0
254	E. M. 6	37.7	2.94	34.8	92.2
325	C. M.	36.8	1.35	35.4	96.3
314	P. F.	36.3	1.26	35.0	96.6
317	K. P.	34.5	1.23	33.3	96.4
300	F. B. 1	34.3	2.16	32.1	93.6
298	E. M. 3	34.1	2.53	31.6	92.6
297	E. M. 2	32.6	1.95	30.6	94.0
348	M. J. 3	31.3	2.06	29.2	93.3
350	H. J. 2	29.1	1.35	27.7	95.4
281	B. W. 4	27.1	3.53	23.6	87.2
245	E. H.	24.5	1.06	23.4	95.7
282	B. W. 5	23.3	2.92	20.4	87.7
Average.....		40.8	2.48	38.3	93.9

TABLE 9.—Fat Retention of Normal Children on a Mixed Diet; Stools Not Normal. (cf. Table 4.)

No.	Case	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake, Retained
277	R. L. 3	52.1	1.18	50.9	97.5
285	B. W. 13	51.9	5.33	46.6	89.8
286	B. W. 14	51.9	3.70	48.2	93.0
327	R. M.	50.5	4.35	46.1	91.4
337	D. K.	46.6	1.31	45.3	97.2
336	R. K.	45.6	1.62	44.0	96.5
301	F. B. 2	43.4	3.53	39.9	92.0
279	B. W. 2	41.6	4.33	37.3	89.7
328	D. D.	41.3	1.63	39.7	96.1
344	K. v. S.	37.7	1.05	36.6	97.3
247	B. M.	35.6	0.71	34.9	98.0
349	H. J. 1	35.5	1.10	34.4	96.9
347	M. J. 2	32.4	4.44	28.0	86.3
339	E. A. 1	32.2	1.51	30.7	95.3
340	L. S.	30.5	1.58	28.9	94.9
346	E. P.	29.5	1.59	27.9	94.7
338	R. N.	23.9	1.43	22.5	94.1
Average.....		40.1	2.38	37.7	94.0
248	E. D.	9.7	4.42	5.3	54.3

two values in each table below 90. This shows how exceedingly good is the fat retention of most children on a mixed diet. It is also noteworthy that the fat loss in constipated stools is no greater than that in normal stools.

The values for intake, excretion and retention in Table 9 show surprisingly little variation either in range or average from those found when the stools were normal or constipated. For percentage of intake retained there are only four values below 90.

In observation 248 the intake was so unusually low that the figures are not included in the average. The diet in this case consisted largely of carbohydrates (67.9 per cent. of the total calories). Although the fat intake was so low, the excretion of fat was as high as that of some children whose intake was five times as great. The stools showed much starch and vegetable waste.

TABLE 10.—COMPARISON OF AVERAGE FAT RETENTION OF INFANTS AND OLDER CHILDREN

	Character of Stool	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake Retained
Infants on usual milk modifications	Constipated	27.1	2.74	24.4	89.9
	Normal	26.7	2.33	24.4	91.3
Children on milk or milk with bread and cereal	Normal or constipated	29.6	2.76	26.8	90.6
Children on mixed diet	Constipated	41.2	2.51	38.7	93.9
	Not normal	40.1	2.38	37.7	94.0
	Normal	40.8	2.48	38.3	93.9

It is to be noted that the acid abnormal stools reported in Table 9 were not watery stools. These very acid stools, however, may possibly be one of the early indications of a serious digestive disturbance, which might end in diarrhea with a high loss of fat in the stools. That an excessive fat loss with consequently lowered retention occurs as a result of diarrhea was clearly shown with our breast fed and artificially fed infants. Very loose stools, however, are not frequently seen with older children on a mixed diet, unless the children are acutely ill, and no cases are included in this study.

Table 10 gives a comparison of the fat excretion and retention of normal infants on milk modifications with the excretion and retention of normal children on a mixed diet.

The most striking point brought out in Table 10 is the uniformity in the daily fat excretion. The average values show little difference whatever the kind of diet or the amount of fat intake. It is surprising that older children with a much higher intake lost practically the same amount of fat as did the infants. This results in a higher percentage retention for the older children. Here again is emphasized the fact

that the retention of fat is the same when the stools were constipated as when they were of normal consistency.

The second part of the paper deals with two groups of children not in normal condition as to digestion. In the first group are considered the rachitic cases; in the second are considered the children suffering from chronic intestinal indigestion, in several cases with intestinal infantilism.

3. FAT CONTENT AND DISTRIBUTION IN STOOLS OF RACHITIC CHILDREN

In Table 11 are given the analyses of the stools which were normal or constipated, and in Table 12 are given the analyses of those which were acid in reaction and showed undigested food or evidence of fermentation.

TABLE 11.—FAT PER CENT. AND DISTRIBUTION IN STOOLS OF RACHITIC CHILDREN ON A MIXED DIET. STOOLS NORMAL OR CONSTIPATED

No.	Case	Age	Diet	Intake of Fat, Grams Daily	Analyses of Stools			
					Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
						Soap	Free Fatty Acids	Neutral Fat
262	F. W. 2	3 yr. 2 mo.	35 oz. milk, meat, etc.	33.8	20.1	72.0	13.3	14.7
316	L. R. 2	1 yr. 11 mo.	40 oz. milk, egg, etc.	52.5	32.0	64.3	12.8	22.9
265	I. H. 1	2 yr. 6 mo.	32 oz. milk, egg, meat, C.L.O., etc.	58.3	30.4	63.2	21.4	15.4
315	L. R. 1	1 yr. 10 mo.	34 oz. milk, egg, etc.	39.8	25.4	62.6	15.5	21.9
261	F. W. 1	3 yr. 1 mo.	30 oz. milk, meat, etc.	24.2	23.7	61.4	22.1	16.5
262	F. W. 3	3 yr. 4 mo.	32 oz. milk, egg, meat, C.L.O., etc.	56.7	32.1	59.6	19.7	20.7
256	R. M. 2	2 yr. 7 mo.	29 oz. milk, eggs, C.L.O., etc.	55.7	50.7	48.8	35.8	15.4
258	R. M. 4	2 yr. 10 mo.	30 oz. milk, egg, olive oil, etc.	47.3	44.9	45.4	25.7	28.8
257	R. M. 3	2 yr. 8 mo.	27 oz. milk, eggs, butter, etc.	61.0	36.9	44.7	26.8	28.5
266	I. H. 2	2 yr. 8 mo.	32 oz. milk, egg, meat, C.L.O., etc.	46.9	20.6	40.6	36.8	22.6
Average				47.6	31.7	56.3	23.0	20.7

The stools considered in Table 11 were all alkaline to litmus, and in Nos. 261, 256 and 258 were constipated. It is noteworthy that the diet in every case contained a large amount of milk, the smallest amount being 27 ounces daily. The average fat intake is higher than that of any group of the normal children on mixed diet because of the addition of cod liver oil or other fat for therapeutic purposes.

The fat percentage of dried weight is distinctly higher than that found for children in normal condition. The soap percentage of total fat is higher and the neutral fat and fatty acids correspondingly lower than in similar stools of normal children. The stools which were constipated show no higher values for soap than do the others.

The stools considered in Table 12 were all acid and showed undigested food, mucus or evidences of fermentation. In this group the amount of milk taken was much less than in the preceding group, the largest amount shown in this table being less than the smallest seen in Table 11. There is a possible connection between the small intake of milk and the acid abnormal stools. This was previously suggested in the discussion of Table 9 of this paper. The fat intake shown in Table 12 averages lower than in Table 11, being about the same as that found for the normal children.

TABLE 12.—FAT PER CENT. AND DISTRIBUTION IN STOOLS OF RACHITIC CHILDREN ON A MIXED DIET. STOOLS NOT NORMAL

No.	Case	Age	Diet	Intake of Fat, Grams Daily	Analyses of Stools			
					Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
						Soap	Free Fatty Acids	Neutral Fat
304	R. A. 1	4 yr.	24 oz. milk, butter, egg, meat, etc.	42.1	23.3	55.5	24.4	20.1
357	R. A. 3	4 yr. 4 mo.	24 oz. milk, butter, egg, etc.	58.1	18.6	54.3	22.9	22.8
356	R. A. 2	4 yr. 3 mo.	24 oz. milk, butter, egg, etc.	51.5	14.8	44.5	26.2	29.3
253	R. M. 1	2 yr. 5 mo.	12 oz. protein milk, 13 oz. milk, cereal	19.6	38.2	43.4	37.3	19.3
235	L. H. 2	1 yr. 6 mo.	26 oz. protein milk, egg, etc.	45.1	32.2	37.8	....	....
333	L. H. 1	1 yr. 5 mo.	19 oz. milk, egg, etc.	33.5	20.6	19.4	....	....
	Average			41.7	24.6	42.5	27.7	22.9

The average fat percentage of dried weight is lower than that found in the normal stools of the rachitic children, but higher than that in the stools of normal children. It has been our observation that in stools containing much mucus or waste the fat percentage of dried weight is always lower than in the corresponding group of normal stools. As compared with the normal stools of rachitic children there is here shown an increase of fatty acids at the expense of soap, which would probably have been more marked if the moist stools had been analyzed, since these stools were all acid in reaction.

4. FAT RETENTION OF RACHITIC CHILDREN

Tables 13 and 14 show the excretion and retention of fat of these same rachitic children. Table 13 gives the values when the stools were normal or constipated, and Table 14 those when the stools were acid abnormal ones.

In Table 13 the average daily fat loss, 3.98 gm., although not excessive, is greater than that of normal children on a corresponding diet. Three very high values for fat excretion (Nos. 256, 257, 258) were

obtained with the same child (R. M.); excluding these, the average differs little from normal. The percentage of fat intake retained is somewhat lower than that found for normal children, largely because of the values found in the case of R. M. The actual retention is higher than that found for the normal children because of the higher intake.

The average values shown in Table 14 for fat loss and percentage retention are not essentially different from those seen in Table 13. The actual retention is less because of the smaller intake.

TABLE 13.—FAT RETENTION OF RACHITIC CHILDREN. STOOLS NORMAL. (CF. TABLE 11.)

No.	Case	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake, Retained
257	R. M. 3	61.0	6.42	54.6	89.6
265	I. H. 1	58.3	1.49	56.8	97.3
263	F. W. 3	56.7	3.44	53.3	94.0
256	R. M. 2	55.7	8.59	47.1	84.6
316	L. R. 2	52.5	3.56	48.9	93.0
258	R. M. 4	47.3	7.34	40.0	84.4
266	I. H. 2	46.9	1.58	45.3	96.7
315	L. R. 1	39.8	1.96	37.8	95.0
262	F. W. 2	33.8	2.49	31.3	92.7
261	F. W. 1	24.2	2.97	21.2	87.8
Average.....		47.6	3.98	43.6	91.6

TABLE 14.—FAT RETENTION OF RACHITIC CHILDREN. STOOLS NOT NORMAL (CF. TABLE 12.)

No.	Case	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake, Retained
357	R. A. 3	58.1	2.33	55.8	96.1
356	R. A. 2	51.5	2.17	49.3	96.0
235	L. H. 2	45.1	5.71	39.4	87.4
304	R. A. 1	42.1	3.61	38.5	91.4
333	L. H. 1	33.5	2.62	30.9	92.3
253	R. M. 1	19.6	4.24	15.4	78 4
Average.....		41.7	3 45	38.2	91.6

The number of cases reported in these tables is too small to permit of any final conclusions, but the findings indicate that rachitic children have a somewhat larger excretion of fat than is usually seen with normal children on a similar diet.

5. FAT PERCENTAGE AND DISTRIBUTION IN STOOLS OF CHILDREN SUFFERING FROM CHRONIC INTESTINAL INDIGESTION

Tables 15 and 16 present the fat percentage and distribution of fat in the stools of the children suffering from chronic intestinal indigestion. These children represent various types of this condition, but they all had habitually large stools in proportion to the amount of food

taken. The observations are divided into two groups according to the character of the stools. The first group is that in which the stools were alkaline or amphoteric. These were usually fairly homogeneous and smooth. All but two (Nos. 341 and 239) were very foul. The other group is that in which the stools were strongly acid, containing mucus — usually in considerable amounts — and showing marked evidences of fermentation.

There was great variety in the diet of the children in these groups. All but one (No. 334) had an unusually low fat provided by milk, either because of a small amount of milk or because the milk was partly or completely skimmed. Several had buttermilk, indicated in the tables as L. A. (lactic acid) milk. In some cases the fat of the diet was increased by the addition of butter or cod liver oil.

TABLE 15.—FAT PER CENT. AND DISTRIBUTION IN STOOLS OF CHILDREN SUFFERING FROM CHRONIC INTESTINAL INDIGESTION; DIET MIXED; STOOLS ALKALINE

No.	Case	Age	Diet	Intake of Fat, Grams Daily	Analyses of Stools			
					Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
						Soap	Free Fatty Acids	Neutral Fat
236	G. R. 1	5 yr.	26 oz. fat-free L. A. Milk, 2 tbsp. cereal with 4 oz. milk	12.7	28.0	71.7	18.2	10.1
237	G. R. 2	5 yr. 8 mo.	9 oz. fat-free L. A. milk, 19 oz. 2% L. A. milk, cereal with 6 oz. milk, egg, etc.	35.8	47.0	70.7	15.6	13.7
341	A. E.	2 yr. 9 mo.	10 oz. milk, butter, etc.	15.5	30.8	70.5	20.4	9.1
242	F. G. 4	6 yr. 5 mo.	13 oz. fat-free L. A. milk, eggs, 16 oz. 2% L. A. milk, meat, C.L.O., etc.	33.0	28.2	56.5	15.5	28.0
241	F. G. 3	6 yr.	20 oz. 2% L. A. milk, nut butter, egg, meat, C.L.O., etc.	58.9	38.7	53.1	22.8	24.1
335	E. R. 1	8 yr. 5 mo.	8 oz. milk, butter, egg, meat, etc.	29.2	60.5	42.1	31.9	26.0
332	N. C. 1	6 yr.	36 oz. skimmed milk, egg, meat, etc.	23.5	21.1	39.8	47.0	13.2
239	G. R. 4	8 yr.	29 oz. 2% L. A. milk, egg, meat, cereal with 9 oz. milk, C.L.O., etc.	55.1	28.5	37.6	28.2	34.2
323	E. R. 2	9 yr. 4 mo.	20 oz. milk, egg, meat, butter, C.L.O., etc.	48.4	46.5	32.3	43.8	23.9
331	N. C. 2	6 yr. 4 mo.	36 oz. skimmed milk, egg, meat, C.L.O., etc.	37.6	34.5	30.5	55.2	14.3
	Average			35.0	36.4	50.5	29.9	19.6

The striking point brought out in Table 15, which gives the findings on the alkaline stools, is the high fat percentage of dried weight. The average, 36.4, is very much higher than that found for any group of normal children on a mixed diet, and only one value in this table even approximates the normal average. The average percentage of soap is somewhat higher than that found in the stools of normal children. This high value is due, however, to three instances in which the amounts of solid food were small. The remaining cases show a range and average similar to that found with normal children. The neutral

fat is in both range and average rather lower than that for normal children, while the fatty acids are correspondingly higher.

The fat percentage of dried weight shown in Table 16, in which are reported the findings on the acid stools, is very high for children on a mixed diet. The average, 35.3, is almost the same as that in Table 15. The chief difference between these findings and those on the alkaline stools of similar children is in the great increase of fatty acids at the expense of soap. This difference would be even greater if the acid stools had been analyzed moist. The neutral fat averages just the

TABLE 16.—FAT PER CENT. AND DISTRIBUTION IN STOOLS OF CHILDREN SUFFERING FROM CHRONIC INTESTINAL INDIGESTION; DIET MIXED; STOOLS ACID

No.	Case	Age	Diet	Intake of Fat, Grams Daily	Analyses of Stools			
					Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
						Soap	Free Fatty Acids	Neutral Fat
244	H. F. 2	3 yr. 3 mo.	16 oz. protein milk, butter, meat, etc.	39.8	21.2	55.6	27.2	17.2
238	G. R. 3	7 yr. 8 mo.	22 oz. 2% L. A. milk, cereal with 9 oz. milk, egg, meat, C.L.O., etc.	49.0	34.8	54.1	19.8	26.1
294	M. H.	2 yr. 1 mo.	19 oz. milk, meat, butter, etc.	30.5	34.6	42.9	48.2	8.9
278	W. R. 1	4 yr. 6 mo.	16 oz. milk, nut butter, egg, etc.	35.2	21.7	42.4	32.4	25.2
255	D. R.	3 yr.	17 oz. skimmed milk, egg, butter, etc.	23.8	42.3	49.6	33.1	17.3
249	S. M.	2 yr. 10 mo.	12 oz. 2% L. A. milk, butter, egg, 12 oz. milk, etc.	36.7	45.1	36.6	45.0	18.4
240	F. G. 2	5 yr. 5 mo.	32 oz. 2% L. A. milk, egg, meat, butter, C.L.O., etc.	74.2	59.7	30.3	55.4	14.3
334	H. L. 1	8 yr.	36 oz. milk, meat, etc.	33.3	30.8	28.3	52.7	19.0
342	F. G. 1	5 yr.	No milk; egg, butter, etc.	21.0	29.8	26.6	33.8	39.6
243	H. F. 1	2 yr. 10 mo.	32 oz. protein milk (low fat), meat, large amount carbohydrates, etc.	17.1	33.3	24.8	65.2	10.0
Average				36.1	35.3	39.1	41.3	19.6
343	H. L. 2	8 yr	6 oz. milk, butter, etc. (much potato)	34.6	19.5	15.6	70.4	14.0

same in both types of stools and suggests a good splitting of the ingested fat, which is rather surprising. The values in No. 343 are not included in the average because the stool was mixed with the urine. The exceptionally low value for fat percentage of dried weight in this case is due to the inclusion of the urine solids in the total dried weight. This illustrates the importance of carefully excluding urine in the collection of stools for analysis. The distribution of fat shown in this case is not unusual for this type of stool, but because of the contamination no reliance can be placed on the figures. The observation is included because the retention values, which are reliable, are reported in a succeeding table.

6. FAT RETENTION OF CHILDREN SUFFERING FROM CHRONIC INTESTINAL INDIGESTION

Tables 17 and 18 give the excretion and retention of fat of the children with chronic intestinal indigestion. Table 17 gives the values found when the stools were alkaline or amphoteric. Table 18 those when the stools were distinctly acid.

TABLE 17.—FAT RETENTION OF CHILDREN SUFFERING FROM CHRONIC INTESTINAL INDIGESTION; STOOLS ALKALINE (CF. TABLE 15)

No.	Case	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake, Retained
241	F. F. 3	58.9	9.54	49.4	83.9
239	G. R. 4	55.1	5.11	50.0	90.8
323	E. R. 2	48.4	14.64	33.8	69.7
331	N. C. 2	37.6	5.99	31.6	84.1
237	G. R. 2	35.8	11.20	24.6	68 8
242	F. G. 4	33.0	6.19	26.8	81.3
535	E. R. 1	29.2	11.20	18.0	61.6
332	N. C. 1	23.5	3.98	19.5	83.2
341	A. E.	15.5	1.95	13.6	87.8
236	G. R. 1	12.7	3.09	9.6	75.7
Average		35.0	7.29	27.7	79.1

TABLE 18.—FAT RETENTION OF CHILDREN SUFFERING FROM CHRONIC INTESTINAL INDIGESTION; STOOLS ACID (CF. TABLE 16)

No.	Case	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake, Retained
240	F. G. 2	74.2	18.67	55.5	74.8
238	G. R. 3	49.0	7.49	41.5	84.8
244	H. F. 2	39.8	3.54	36.3	91.1
249	S. M.	36.7	7.30	29.4	80.2
278	W. R. 1	35.2	4.34	30.9	87.8
243	H. L. 2	34.6	8.95	25.6	74.0
334	H. L. 1	33.3	11.34	22.0	66.1
294	M. H.	30.5	4.79	25.7	84.3
255	D. R.	25.8	4.68	19.1	80.2
242	F. G. 1	21.0	4.68	16.3	77.6
243	H. F. 1	17.1	12.50	4.6	26.5
Average		35.9	8.03	27.9	77.7

There is no essential difference in fat excretion and retention whether the stools were alkaline or acid. In both tables is shown a very great loss of fat, the averages being, respectively, 7.29 and 8.03 gm. daily, which is about three times the normal average for children on mixed diet. In six instances the daily fat loss exceeds 10 gm. In only one instance (No. 341) is the loss less than 3.0 gm. This child, A. E., was very small and at the time was taking very little food.

The average percentage of fat retained is 79.1 for the group with alkaline stools and 77.7 for the group with acid stools. With only one of the children referred to in each group is the fat retained as much

as 90 per cent. of the fat intake. In the group where the stools were alkaline, a retention of 90.8 per cent. is seen in the case of G. R. 4 (No. 239). The condition of this child, although previously very serious, was greatly improved at the time this observation was made. In the group where the stools were acid there is in the case of H. F. 2 (No. 244) a retention of 91.1 per cent. of the intake. This child had since the first observation (cf. H. F. 1, No. 243) been put on a diet in which the fat was increased from 19 to 33 per cent. of the total calories, with the result that the total fat intake was more than doubled. It is interesting to note that this child with a chronic and very serious indigestion shows with increased fat intake the highest retention in the group, notwithstanding the fact that the stools were still acid. This suggests that the trouble in this case was more with carbohydrates than with fat.

TABLE 19.—Comparison of Average Analyses of Stools of Normal Children and Those Suffering from Rickets and from Chronic Intestinal Indigestion

Condition of Child	Character of Stools	Intake of Fat, Grams Daily	Analyses of Stools			
			Fat per Cent. of Dried Weight	Per Cent. of Total Fat as		
				Soap	Free Fatty Acids	Neutral Fat
Normal	Normal—alkaline	40.5	18.0	45.1	27.4	27.5
	Not normal—acid	38.4	15.1	32.8	31.3	35.9
Rachitic	Normal—alkaline	47.6	31.7	56.3	23 0	20.7
	Not normal—acid	41.7	24.6	42.5	27.7	22.9
With chronic intestinal indigestion	Normal—alkaline	35.0	36.4	50.5	29.9	19.6
	Not normal—acid	36.1	35.3	39.1	41.3	19.6

The actual fat retention of the children with chronic intestinal indigestion varies greatly in the different cases, but in those in which the intake was very high the retention was also high, being quite equal to the normal average. This may have an important clinical bearing. The fact that the fat excretion is abnormally high is not in itself a sufficient reason for cutting down the fat in the diet. The really important thing as far as fat metabolism is concerned is the amount of fat retained.

Table 19 gives a comparison of the average analyses of the stools of normal children and those suffering from rickets and from chronic intestinal indigestion.

It is shown in Table 19 that with all three types of children studied the fat percentage of dried weight is lower in the acid stools than in the normal alkaline stools. The fat percentage of dried weight is much higher in the stools of the abnormal children than in those of the

normal children, and is greater in those of the children suffering from chronic intestinal indigestion than in those of the children suffering from rickets.

In each of the groups shown in Table 19 the soap percentage is lower in the acid than in the alkaline stools. In each group also the percentage of fatty acids is greater in the acid stools than in the alkaline, the difference being most marked with children suffering from chronic intestinal indigestion. With normal children the percentage of neutral fat is much higher in the acid stools than in the alkaline, but with abnormal children there is little or no difference in percentage of neutral fat whether the stools were acid or alkaline. The stools of the normal children show a somewhat higher neutral fat and lower soap than do those of the abnormal children. This may not be significant because of the wide range of values shown for the abnormal children and because there was such a wide variety in the diet.

Table 20 compares the average excretion and retention of fat by the normal children and the abnormal children.

TABLE 20.—COMPARISON OF AVERAGE RETENTION OF FAT OF NORMAL CHILDREN AND OF THOSE SUFFERING FROM RICKETS AND FROM CHRONIC INTESTINAL INDIGESTION

Condition of Child	Character of Stools	Intake of Fat, Gm. Daily	Fat in Stools, Gm. Daily	Fat Retained, Gm. Daily	Per Cent. of Intake, Retained
Normal	Normal, alkaline	40.8	2.48	38.3	93.9
	Not normal, acid	40.1	2.38	37.7	94.0
Rachitic	Normal, alkaline	47.6	3.98	43.6	91.6
	Not normal, acid	41.7	3.45	38.2	91.6
With chronic intestinal indigestion	Normal, alkaline	35.0	7.29	27.7	79.1
	Not normal, acid	35.9	8.03	27.9	77.7

Table 20 brings out more clearly the points emphasized in the discussion of the separate retention tables. With all three types of children there is no significant difference in fat excretion or retention, whether the stools were normal alkaline or abnormal acid ones. The fat loss in the stools of the two types of abnormal is greater than that of normal children, being somewhat increased with rachitic children and very greatly increased with those suffering from chronic intestinal indigestion. The percentage of the fat intake retained by the rachitic children is but little lower than is usually found in normal cases because of the high intake and because the loss in the stools, although greater than in normal cases, is not excessive. The percentage retention of fat of the children suffering from chronic intestinal indigestion, however, is much lower than is that of normal children, owing to the abnormally large excretion.

SUMMARY

1. In the normal or constipated stools of older children whose diet consisted of milk alone or milk with bread and cereal the fat percentage of dried weight averaged 30.7, which is lower than the average found for similar stools of infants taking modifications of cow's milk. The soap percentage of total fat averaged 60.9, which was somewhat lower than that found in the stools of the infants.

2. The normal and the constipated stools of children on a mixed diet showed almost identical average values both for fat percentage of dried weight and for distribution of fat. The fat percentage of dried weight averaged, respectively, 18.0 and 20.1, and the soap averaged, respectively, 45.1 and 47.9 per cent. of the total fat. These values were much lower than those found when the diet contained little or no solid food.

3. In the acid abnormal stools of children on a mixed diet the fat averaged 15.1 per cent. of the dried weight. Both the fat percentage of dried weight and the soap percentage of total fat were much lower than in normal stools and the values for fatty acids and for neutral fat were higher.

4. With rachitic children the fat percentage of dried weight averaged 34.7 in the alkaline stools, and 24.6 in the acid stools. The values were higher than those found for corresponding types of stools of normal children. The proportions of soap, fatty acids and neutral fat were not significantly different from those for normal children.

5. The stools of children suffering from chronic intestinal indigestion showed a much higher fat percentage of dried weight than those of normal children; the average for alkaline stools being 36.4 per cent., and for acid stools 35.3 per cent. The average percentage of neutral fat was lower in both alkaline and acid stools of these children than in the stools of normal children. The fatty acids were higher than normal, much higher when the reaction of the stools was acid.

6. The average fat loss in the stools of normal children varied between 2.6 and 3.0 gm. in all the groups studied, being highest in the stools of children whose diet contained the smallest proportion of solid food and the largest proportion of milk.

7. The normal children on mixed diet retained on the average about 94 per cent. of the fat intake, regardless of the type of stool. The average actual retention was about 38 gm. daily. The children with little or no solid food and a smaller fat intake showed a lower actual and a somewhat lower percentage retention than those on a general mixed diet.

8. The rachitic children showed a slightly larger fat loss in the stools than did the normal children; their intake, however, was higher. Their actual retention, therefore, equalled or exceeded that of the normal children, and their percentage retention was only a little lower than the normal average.

9. The fat loss in the stools of the children suffering from chronic intestinal indigestion was very great, averaging 7.3 gm. daily in the alkaline stools and 8.0 gm. in the acid stools. Both the actual and percentage retention were much lower than normal. The percentage of the intake retained averaged 79.1 when the stools were alkaline and 77.7 when they were acid. When the intake of fat was very high the actual retention was usually as high as that found for normal children

A CLASSICAL CASE OF HEMOPHILIC ARTHRITIS*

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CASE—William B., 9½ years of age, born in Jersey City, N. J., of Italian parentage, has been under observation at the Cornell Children's Clinic for the past two years.

Family History.—His paternal history, as far as known, is free from hemophilia. On his mother's side the disease can be traced back two generations. His maternal grandmother died of tuberculosis. Whether she had any symptoms of hemophilia is doubtful. She had twelve children, several of whom died of tuberculosis. A history was obtained of only six of these children, two sons and four daughters. One of the boys died of tuberculosis. He was not considered a bleeder. The other died of hemorrhage when 18 years of age. He was a bleeder. One of the girls gave a history of purpuric spots, spontaneous ecchymosis and bruises following trauma from childhood. Of her offspring, two children, a boy and a girl, are not considered bleeders. The second girl had three children, boys, two of whom are bleeders. The third girl had eight boys, seven of whom are bleeders. One boy is normal. The fourth girl, the patient's mother, gives a similar personal history. She had twelve children and several induced miscarriages. Of eight boys and four girls, four boys and three girls are living. One boy died at 2 years of age following a convulsion. He was a bleeder. The other children died in infancy and were not considered bleeders.

The patient's sisters, aged 20, 16 and 7 years, respectively, all give a history of purpuric spots and bruises; no other symptoms. Two of his brothers, 13 and 17 years of age, respectively, give no evidence of hemophilia. The third brother, 24 years of age, gives a typical history. At birth a purpuric spot was noted on his face. He had frequent hemorrhages, spontaneous and following trauma. They were noted early. He had hematuria and epistaxis when 5 years of age; then followed the joint involvement at the age of 8. Frequent severe repeated attacks, involving the knee, ankle and elbow joints, left him a cripple, confined to bed almost constantly, until puberty, when the interval between attacks of hemarthrosis increased and the severity of the symptoms gradually diminished. For the past three years he has been free from joint involvement, but he has had some joint pains and ecchymosis. He still has some deformity and some stiffness of left knee, ankle and elbow (Figs. 5 and 6). It is interesting to note that he was accepted for limited military service, and in spite of a week of trench digging and hiking he did not have any return of symptoms. The patient is the fourth of these boys.

Summary.—In summing up the family history the following points are of special interest.

(1) The definite maternal transmission of hemophilia to the majority of the males through two generations. (2) The persistent evident hereditary purpuric tendency in the females. (3) The presence of typical hemophilia with arthritis in the brother, with recrudescence of symptoms after puberty. (4) The characteristic marked fertility and larger proportion of males in the offspring. (5) The history of tuberculosis.

General Physical Examination.—The patient is malnourished, anemic, dark complexioned with dark hair and eyes, of slender build, good intelligence and

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Fig. 1.—There is marked swelling of the left knee joint, slight swelling of the right elbow and thickening of the second phalangeal joint of the index finger of the right hand.

Fig. 2.—Lateral view of left knee joint. Note the characteristic posture.

Fig. 3.—Anteroposterior and lateral views of the left elbow, showing considerable enlargement of the centers of ossification for the head of the radius and external condyle. The center for the olecranon has not only appeared but has united. In the print of the right elbow this center has not appeared. It is seen normally at about the eighth year and unites with the shaft about the sixteenth year.

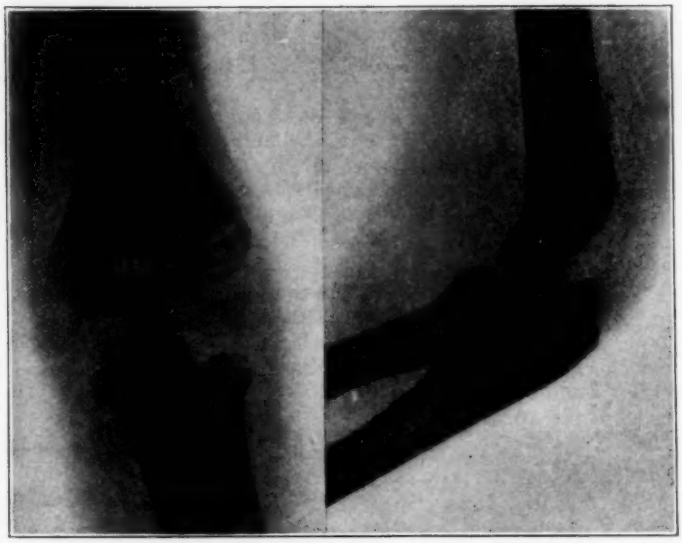


Fig. 4.—Anteroposterior and lateral views of the right elbow joint, apparently normal now; at least there is no marked roentgen-ray evidence that this joint is or has been involved.

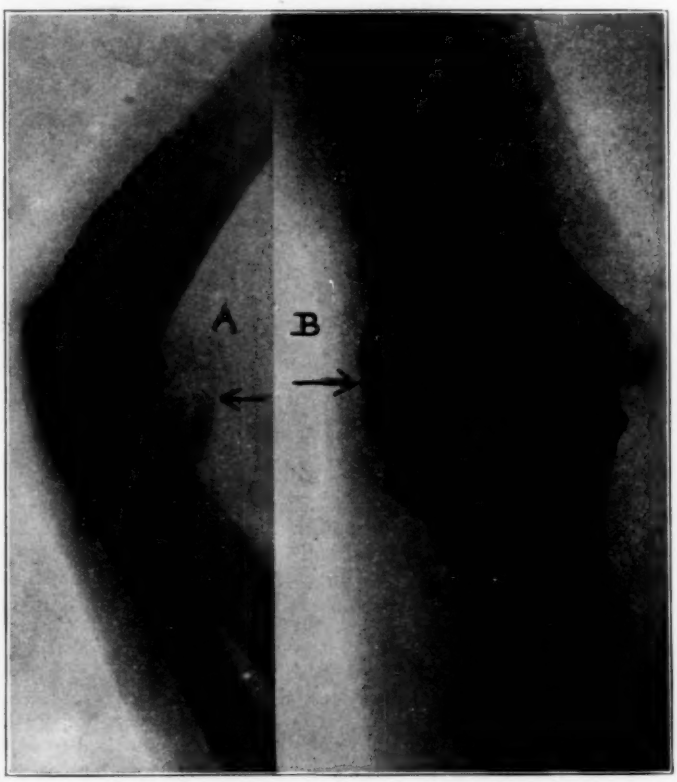


Fig. 5.—(a) Lateral view of left elbow of John B. (brother), 24 years of age, showing marked hypertrophy or enlargement due to an increase in lime salt deposit in the coronoid process of the ulna. (b) The anteroposterior view shows a similar condition present in the external condyle.

sanguine temperament, corresponding to the erethetic type of Wachmuth.[1] The throat, teeth, heart and lungs are normal. D'Espine's sign is present to the fourth dorsal vertebra. The liver and spleen are normal.

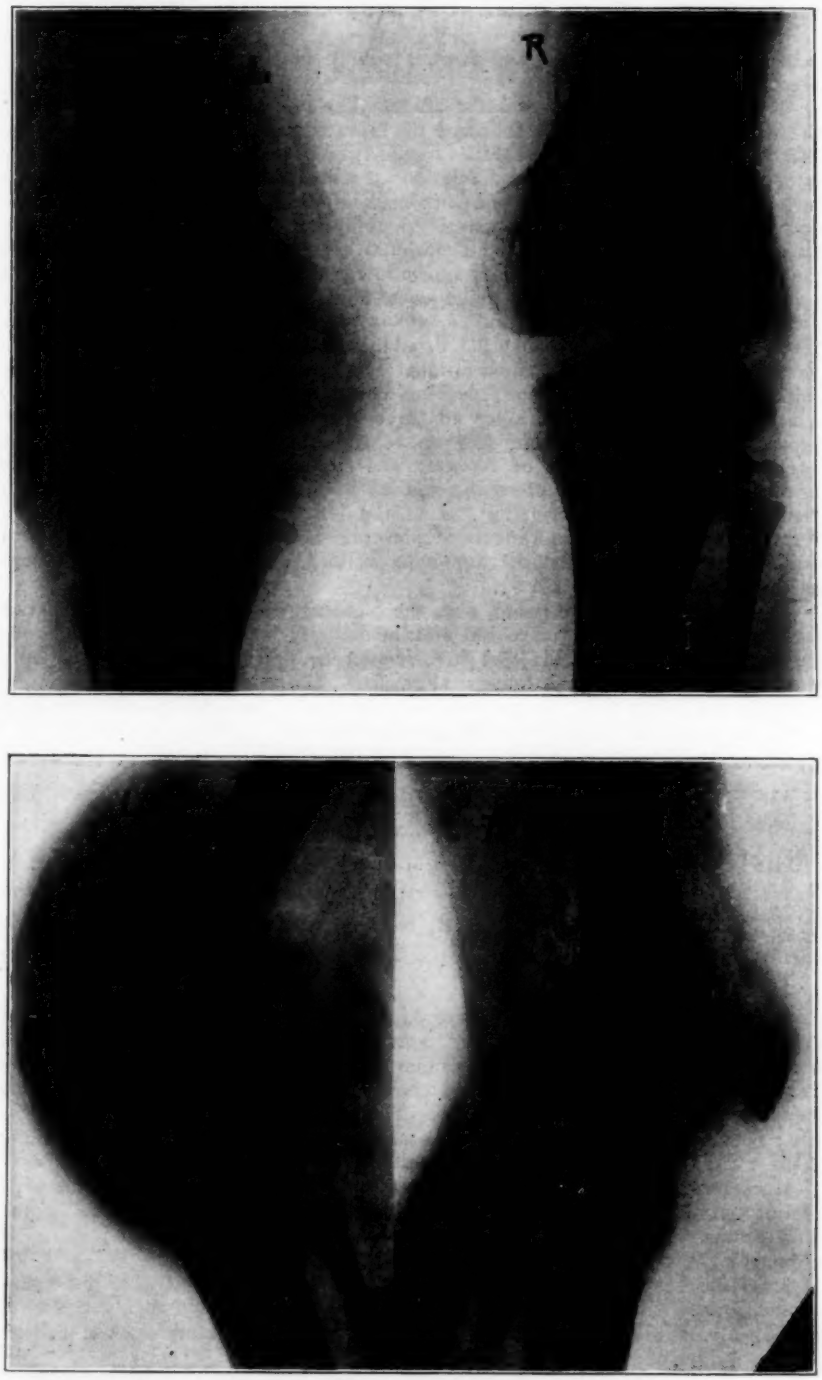
Personal History.—Although a normal birth, the boy was breast fed for two months. He had measles, scarlet fever and pertussis without any untoward symptoms or complications. He is subject to repeated attacks of bronchitis. Vaccination was not attended with any bleeding.

Hemophiliac History.—After birth a purple spot, the size of a quarter, was found on the palm of the right hand. This spot disappeared in a short time. (The mother claims that every male bleeder in the family showed some such spot at birth.) There was persistent oozing, not of great amount, from the umbilicus for one month. At 2 years of age the spots or spontaneous hem-

Fig. 6.—(a) The anteroposterior and lateral views of the left knee joint of John B. (brother) reveal the presence of spurlike projections and enlargement or elongation of the patella. (b) The anteroposterior view resembles a chronic hypertrophic osteoarthritic joint. It looks like an old man's knee joint.

orrhages under the skin and from the mucous membranes appeared. Slight trauma was followed by bleeding and bruises. When he was 3 years of age epistaxis almost proved fatal. First dentition was normal; second dentition was associated with constant oozing around the gums for several months. There is no history of hematuria. At 4 years of age he had repeated convulsions for two weeks, with a hemiplegia which cleared up. This is suggestive of a cortical hemorrhage. Recently a scratch of the tongue with a crust of bread caused bleeding that was controlled with difficulty. Ten days ago a

1. Legg, Wickham: Treatise on Hemophilia, London, 1872, p. 46.



Figs. 7 and 8.—Evidence of fluid is observed. The joint is hazy; the epiphyses present a hazy, irregular outline. Anteroposterior and lateral views of both knee joints show some enlargement of the epiphyses and the patella, the right being smaller. The lateral views show best the fibrous deposit along the joint side of the epiphyses of the femora. The process is a productive one, there being no evidence of destruction of either cartilage or bone.

hematoma, the size of a grape fruit, appeared in the left groin. It is now absorbed.

The first joint involved was the ankle. The patient was 5 years of age at the time. This attack was followed by repeated attacks involving the elbow and knee joint. Occasionally the smaller joints were affected at irregular intervals without any particular relation to climate or weather. There seems to be a definite history of trauma with many of the attacks. The usual course and symptoms of the hemarthrosis is as follows: Either following a trauma or spontaneous and preceded by prodromal symptoms such as acute pains in limbs and joints, facial pallor and anorexia for a few days, one or more joints suddenly become swollen and filled with fluid. They were hot, tender, not red, stiff and extremely painful. Associated with the local symptoms was fever varying between 102 and 103 F. and extreme prostration. After a week or ten days the local and constitutional symptoms abate, the effusion in the joints either is absorbed entirely, leaving a normal joint (as in the right elbow, which ten days ago was swollen to three times its size and is now normal except for a black and blue spot above the internal condyle, and a slight swelling), or as the result of repeated attacks there is left some stiffness as in the left elbow; or it remains the seat of a subacute or chronic process with distinct deformity and crippling, as in the left knee. This joint has been for the past three months (and is at present) swollen, painful and filled with fluid. No doubt the constant trauma in walking due to the deformity keeps up the effusion (Figs. 7 and 8).

Summing up there are noted, first, the bleeding, spontaneous and traumatic; second, the ecchymosis, petechiae and hematoma, both traumatic and spontaneous; third, the typical joint involvement as described by Franz Hoenig and illustrative of the pathology (Fig. 5).[2] (1) Hemarthrosis, effusion of blood in the joint; (2) subsequent attacks producing panarthritis and (3) end-result deformity and ankylosis (Figs. 6 and 7).

Blood Examination.—A careful blood study was made by A. F. Hess[3] two years ago and showed the following:

Date	Coagulation Time Plasma, Minutes	Coagulation Time Blood, Minutes	Remarks
11/13/14.....	52	90	
11/20/14.....	20	55	
11/24/14.....	...	106	
12/16/14.....	100	210	(Five days ago severe bleeding from tongue)
1/14/15.....	24		

Plasma of brother, M. B., 9 years of age, clotted in nine minutes. Plasma of brother, John B., clotted in twenty-six minutes. A recent test made at the base hospital showed clotting in fifteen minutes. Besides the marked delay in coagulation time there was diminished prothrombin content. The platelets were normal, from 200,000 to 300,000. The capillary resistance test was negative; puncture test was negative. The antithrombin test was normal. Adding calcium did not change the coagulation time.

Roentgen-ray examination showed the process to be one of production of fibrous tissue in the joints subject to repeated attacks, as can be seen in the illustrations. The hemarthrosis may clear up and leave a normal joint as is shown by the roentgenogram of the right elbow.

I am indebted to Dr. R. E. Herendeen from the department of roentgenology, Cornell University Medical College, for the excellent roentgenograms, and to Mr. William Dunn from the photographic laboratory of the college for the prints and photographs.

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2. Bulloch and Fildes: Hemophilia, Eugenic Lab. Memoirs, Univ. of London, 1911, 12.

3. Hess, Alfred F.: The Blood Vessels in Hemophilia and Other Hemorrhagic Diseases, Arch. Int. Med. **17**:203 (Feb.) 1916.

PROGRESS IN PEDIATRICS

REVIEW OF RECENT LITERATURE ON THE NEW-BORN

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CHILDREN'S CLINIC OF MINNEAPOLIS

OPHTHALMIA NEONATORUM

In his review of the effect of venereal disease on infant mortality, Bartlett[93] says that 70 per cent. of ophthalmia neonatorum is due to gonorrhea.

Tivnen[94] in an article covering the scope of blindness from ophthalmia neonatorum states that one-eighth of all blindness and one-fourth of blindness among children is attributable to this disease, all of which could have been avoided by a little attention at the right time. Cases occurring before the third day are almost always due to gonococcus infection. He advocates 1 drop of 2 per cent. silver nitrate solution in the eye to be used routinely as a prophylactic measure.

Thompson[95] states that there is a great difference in the disease in the infant and the adult and the mother probably confers a strong degree of immunity on the child. Infection ordinarily starts from the second to the fifth day after birth. Cases occurring before the second day are due to intrauterine infection and those occurring after the tenth day are due to secondary infection. Ulceration always means a mixed infection, and complications may be the same as those from gonococcus infection elsewhere. In this excellent article he outlines the treatment with great detail.

In discussing the best method of caring for patients suffering from ophthalmia neonatorum, Derby[96] favors hospital treatment, the scientific care and feeding so obtained being of sufficient benefit to offset partially the handicap of the ocular trouble. The grave question of the frequent handling necessary for medication every half hour should be governed by the general condition of the infant. If the child is losing in weight, the eye treatment should be decreased, even to one

93. Bartlett, F. H.: Effect of Venereal Disease on Infant Mortality, Am. J. Syphilis **2**:156 (Jan.) 1918.
94. Tiven, R. J.: Blindness Caused by Ophthalmia Neonatorum, J. A. M. A. **63**:1756 (Nov. 12) 1914.
95. Thompson, G. H.: Ophthalmia Neonatorum, Boston M. & S. J. **174**:745 (May 5) 1916.
96. Derby, G. S.: Some Aspects of Ophthalmia Neonatorum, J. A. M. A. **65**:1317 (Oct. 16) 1915.

treatment every three or four hours. Prematurity appears to predispose to the infection. Of the writer's seventy-seven cases due to the gonococcus, 23.4 per cent. were monocular. By placing the patient on the infected side, with the hands bound down, and employing rigid cleanliness, infection in the other eye may be avoided.

By smear and cultural findings of 146 cases, the author states that in 51.3 per cent. of cases, gonococci were found, only three cases of *Micrococcus catarrhalis,* and fifty-six cases in which no organism could be found, probably due to earlier treatment of the cases.

Derby believes that the mildness of the ocular disease in infants is explained by a certain degree of immunity, being transferred by the gonorrheal mother to her offspring. Attempts are being made in Massachusetts to place the responsibility, when an infant suffers the loss of sight, and some kind of prosecution enforced. For local treatment, wash the eye with boric acid, and instill a 25 per cent. solution of argyrol. He believes a mild remedy is indicated in the acute stage of the disease to avoid further reaction.

DeForest,[97] in his very excellent and complete article, presents a comprehensive history of ophthalmia neonatorum and emphasizes the importance of routine instillation of 2 per cent. silver nitrate solution. He goes into his method of treatment and care of the disease when present and calls attention to the better methods for publicity work regarding this infection.

Brav[98] discusses the value of the various new social movements for the eradication of the so-called social disease. He does not believe that ophthalmia neonatorum will be lessened appreciably by any of these movements. He outlines briefly the prophylactic and remedial measures at our disposal for its treatment. Routine use of 2 per cent. nitrate solution as a prophylactic measure is strongly urged; also the use of cold applications, irrigations with 1 to 10,000 bichlorid of mercury solution at one-half hour intervals, 25 per cent. organic silver salt, after irrigation, and painting the conjunctiva once daily with silver nitrate. After a week substitute zinc sulphate solution, one grain to the ounce, for the bichlorid of mercury, and irrigate only every two or three hours. Great care must be used not to injure the cornea, and treatment must be carried on persistently after symptoms clear up.

In spite of legislation toward that end, ophthalmia neonatorum in England does not seem to have been appreciably lessened. Stephen-

97. DeForest, H. P.: Blindness of New-Born, New York M. J. **101:** 1089 (May 29) 1915.

98. Brav, A.: Ophthalmia Neonatorum, Prevention and Curative Treatment, New York M. J. **99:**1029 (May 23) 1914.

son[99] says that 10 per cent. of all blindness is due to this disease, and 30 per cent. of the inmates of institutions for the blind are there as a result of the infection. Midwives are required by law to use prophylactic measures for the prevention of the disease, but the physician is under no such legal obligation, the result being that there is a larger instance of the infection in cases attended by physicians than by the midwives. According to this author, this is true of the United States as well as England. One per cent. silver nitrate solution instilled into the eyes directly after birth is the favorite preventive measure. Scrupulous cleanliness as to the child's eyelids and knuckles is essential. In a study of 1,829 cases, the presence of the gonococcus was determined in 64.65 per cent. He quotes Wharton as finding the gonococcus in 75 per cent. of his cases, Gade in 64 per cent. and Stadfelt in 54.65 per cent. The gonococcus may persist long after the symptoms of ophthalmia have subsided. In fifteen cases the micro-organisms disappeared in from two to seven days before secretion ceased, while in eight cases they persisted for from one to twenty-five days, and have been demonstrated in smears taken sixty days after the disease commenced.

Of every hundred cases of ophthalmia neonatorum, 65 per cent. are due to gonococci, 10 per cent. are due to pneumococci, 5 per cent. to *B. coli,* and 5 per cent. to other organisms, of which, perhaps, the most important are the *B. pyogenes* and the Koch-Weeks bacillus. Many cases of ophthalmia are to be regarded as premonitory or concomitant signs of congenital syphilis.

Infection most commonly arises soon after birth of the head, by the infant blinking into its conjunctival sac the infective material that clings about the eyelids and eyelashes or by rubbing it in with its own knuckle. Antepartum infection is by no means rare, the author having met with nineteen cases. He quotes Haussman and Hellendall in evidence of the possibility of the gonococcus passing through the chorion and amnion, thus reaching the liquor amnii. Ophthalmia that begins on the seventh day is due to secondary infection, while one that commences within twenty-four hours of birth is due to intrauterine infection. Various skin lesions may result from inoculation with the gonococcus during birth. Mammary abscess has been known in the mother nursing an ophthalmic baby.

In the treatment of ophthalmia neonatorum, the basic principle is in the removal of every trace of pus as soon as it is formed. The agents employed are weak antiseptics. The favorites are boric acid, potassium permanganate (1 : 5,000), or corrosive sublimate (1 : 10,000).

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99. Stephenson, S.: Ophthalmia Neonatorum, Practitioner **93**:374 (Sept.) 1914.

The author, however, seldom uses anything more drastic than saline lotion (1.4 per cent.), 25 per cent. argyrol, dropped in the eyes every four hours, and in some cases the conjunctiva is painted with 2 per cent. silver nitrate solution once in twenty-four hours. The value of antigonococcic vaccine is questionable. Another useful remedy is liquid paraffin, with which the conjunctival sac may be filled in intervals between the saline irrigations.

SYPHILIS IN THE NEW-BORN

All offspring of parents who have or have had syphilis should be considered syphilitic until proved otherwise. Read[100] strongly urges a most careful examination of all new-borns as to the existence of a luetic condition at birth. Blood examinations should be made of both child and parents. If these prove negative the child should be treated and tested again as a provocative injection of arsphenamin or mercury may turn the blood test from negative to positive. Watch for snuffles, hoarse cry, rash or marasmus. The latter is of special significance in a breast fed baby showing no signs of indigestion. Peeling of the palms and soles, onychia, thinning of the eyebrows, or any eye lesion is suspicious. Involvement of the liver, spleen, testicles, bones, eyes, nails and other tissues, are danger signs. An unexplained nephritis, a paroxysmal hemoglobinuria, occurring under one year, epiphysitis, within the first three months, marked by acute pain on motion, and apparent paralysis of the limbs may be due to the spirochete in the affected tissue. The author argues against treating the external clinical lesion in children and then waiting in the hope of the cure being complete, only to be met with grave tertiary manifestations years later. Long continued systematic medication is as necessary as in adults, and the cooperation of the parents must be secured. He cites three cases of interstitial keratitis, in two of which early treatment would have prevented partial blindness. Mercury in the form of gray powder, gr. $\frac{1}{2}$ to gr. $\frac{3}{4}$, three times a day, combined with sodium bicarbonate and aromatic powder may be given over long periods. Liquor hydrargyri perchloridi, mm. 10, three times a day, combined with castor oil, mm. 5, and tincture of opium, mm. $\frac{1}{2}$, is a good combination. In gravely progressive lesions he recommends muscular injections of mercury biniodid in aqueous solutions, gr. $\frac{1}{30}$ to gr. $\frac{1}{200}$, twice a week, for three to six weeks. In congenital syphilis, tertiary lesions at an early age require potassium iodid as for adults, 10 to 30 gr., three times a day, for a child of 5 years of age. In all stages the administration of arsphenamin, gr. $\frac{1}{10}$, intravenously is of great value

100. Read, J. S.: Necessity for Early Diagnosis and Continuous Treatment in Congenital Syphilis, Arch. Pediat. **33**:441 (June) 1916.

for a child two years of age. This dose is a safe standard. Read believes that the so-called passive immunity is due to the actual presence of *S. pallidum* in the tissues and is really a latent infection. Treatment must be continued over long periods in childhood, the practitioner being guided not by symptoms alone, but by clinical signs, serology anl all other tests.

The manner in which syphilis in one or both parents affects the offspring is outlined by Haberman[101] as follows: 1. When the organism is transmitted to the embryo and circulates in its blood, resulting in: (*a*) Abortion; (*b*) premature birth; (*c*) still birth; (*d*) the child born at term and lives, but with symptoms of the disease transient or flourishing; (*e*) the child born without symptoms, the secondaries breaking out a few weeks later; (*f*) late hereditary syphilis, appearing in from eight to twenty-eight years later in the tertiary stage. 2. When parents are constitutionally affected and the germ cells are depleted, invalidated or of low vitality resulting in an uninfected embryo, but with almost as sinister a fate, as premature birth or deformities, marasmic and weak children and those easily infected by other diseases. 3. When parents bear perfectly healthy children or some are affected and some are normal. In examining these children, if there is no history of primary, secondary or tertiary symptoms in the parents, have the Wassermann reaction made on both patient and parents. Among the signs to be encountered, aside from idiocy and imbecility, are arrested mentality at the tenth, eleventh, or twelfth year or epileptic seizures. The signs of the disease in the nervous system are apt to develop at puberty. Migraine, chronic vomiting, dizzy spells or persistent chorea may be due to inherited syphilis, also neurasthenia and hysteria, various speech defects or impaired hearing, and sometimes an impaired moral sense. The clinician should also look for the saddle nose, Hutchinson's teeth, rhagades, stiffened knee joints, enlarged spleen and liver, while at times the only suspicious sign may be under development, irritability, sleeplessness and restlessness, or inability to concentrate.

Abner Post[102] discusses the clinical course and physical signs of syphilis in the new-born and defines hereditary syphilis as those cases in which the parents, one or both, were syphilitic at the time of conception, and congenital syphilis as those cases in which the mother was infected during pregnancy. Syphilitic children are born when either or both parents are syphilitic. A pregnant woman who becomes

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101. Haberman, J. V.: Hereditary Syphilis, J. A. M. A. **64**:1141 (April 3) 1915.

102. Post, A.: Clinical Course and Physical Signs in Hereditary Syphilis of Early Age, Am. J. Dis. Child. **12**:364 (Oct.) 1916.

syphilitic transmits the disease to her fetus. A syphilitic baby may suckle its apparently healthy mother with impunity and likewise a syphilitic mother may nurse her apparently healthy baby, born while she was syphilitic, with impunity. Children having early malignant syphilis are usually premature, emaciated, develop snuffles and a hoarse cry. Bullae appear on the palms, soles, wrists and ankles. The lips are cracked and ulcerated. Crusts form at the nostrils, the liver and spleen are enlarged, and the digestion is usually imperfect. They cry at night as if in pain, and have difficulty in nursing because of the condition of the nose. Roentgenologic study of the bones is of some value.

In a comparative study of the luetin and Wassermann reactions for detecting early syphilis, DeBuys and Langford[103] conclude that the Wassermann reaction is not as reliable as the luetin reaction in cases of hereditary syphilis; also that it is impossible for a mother to give birth to a child who gives positive laboratory tests for congenital syphilis without herself giving positive tests. The Wassermann reaction gives evidence of an active process, while the luetin not only gives this evidence, but also indicates an existing syphilitic condition even though it be inactive. The luetin test, however, is difficult to read and slow at development.

In Bartholomew's[104] series of 1,500 cases, there were forty-eight still births, or thirty-two per thousand. Wassermann tests of the mothers were made in only seventeen cases. Of those in which a diagnosis of syphilis was made, the Wassermann reaction was obtained in eight cases, only three of which were positive. Spirochetes were demonstrated in two of the cases, and two of the placentas were syphilitic. In 77 per cent. of the syphilitic cases the placenta showed positive findings, and 75 per cent. of the babies in these cases showed positive findings at necropsy. Sixty to eighty per cent. of macerated, stillborn babies are syphilitic. Examination of the fetal liver is of more value than that of the placenta. Fully one-third of all stillborn children are syphilitic, and 90 per cent. of the children of untreated syphilitic mothers are infected, while 82 per cent. born of the mothers who receive treatment only prior to pregnancy were syphilitic, and 45 per cent. of those children whose mothers were treated during pregnancy.

Roddy[105] reviews the literature on the Wassermann reaction in

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103. DeBuys, R. L., and Langford, J. A.: A Comparative Study of Luetin and Wassermann Reactions in Infancy and Childhood, Am. J. Dis. Child. **12**:387 (Oct.) 1916.

104. Bartholomew, R. A.: Syphilis as the Cause of Still-Birth, J. A. M. A. **70**:289 (Feb. 2) 1918.

105. Roddy, J. A.: Wassermann Test in Congenital Syphilis, Arch. Pediat. **32**:522 (July) 1915.

congenital syphilis and states that after the first year of life patients having active congenital syphilis practically always gave a moderately strong positive Wassermann reaction. The test is not as accurate during the first three months of life as later. The luetin test is said to be preferable to the Wassermann test during the first year of life. He believes that a syphilitic mother may give birth to a healthy child, and that a healthy mother may give birth to a syphilitic child, but in the majority of cases both child and mother are syphilitic. Of twenty infants that gave a positive Wassermann reaction at birth, four became negative in a short time, and of twenty-seven infants that gave a negative reaction at birth five later became positive and two died presenting typical lesions at postmortem.

In true congenital syphilis Blechmann[106] believes that the Noguchi test has seemed less sensitive than the Wassermann test when made with a human antigen. It is not proper to take into consideration a positive luetin test when the clinical and serologic tests are negative, while a positive Wassermann at two different periods in a doubtful specific case should always aid in diagnosis. When negative, the luetin test does not exclude syphilis. The Noguchi test brings to the diagnosis of hereditary syphilis only a secondary complementary information. It is very inferior to the Wassermann test.

Gordon[107] in his report of the luetin reaction in congenital syphilis says that he did not obtain any positive results in children under six months of age, the minimum age for positive reaction being twelve months, although he observed that others have obtained positive reaction even during the first month.

Yerington[108] summarizes the routine work with Wassermann reactions done at the medical department of Stanford University since 1912. The mother's blood was examined, a sample of the baby's blood from the cut cord, and again on the sixth or the tenth day, the child's blood from the heel. A macroscopic and microscopic examination of each placenta was also made. He concludes that a diagnosis of syphilis in an infant under ten days of age cannot be made by a positive cord or heel blood examination alone without evidence of lues elsewhere. The results obtained from examination of heel blood are the more reliable of the two, and a positive heel blood should be considered a danger sign. The investigation of the placenta seems to show

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106. Blechmann, G., Delort, M., and Tulasni, A.: Comparative Results of the Luetin Reaction and the Wassermann in Hereditary Syphilis, Arch. de méd. d. enf. **18**:220 (April) 1915.

107. Gordon, M. B.: Value of Luetin Reaction in Congenital Syphilis, Arch. Pediat. **33**:186 (March) 1916.

108. Yerington, H. H.: Value of Wassermann Reaction in the New-Born, Arch. Pediat. **35**:40 (Jan.) 1918.

that a mother with a negative reaction may give birth to a child stillborn or abortioned because of luetic infection. An attempt on the part of the author is being made to follow up the children who have a positive heel reaction and repeat the Wassermann test to verify the earlier work, and to give treatment where indicated.

Menten[109] believes that no woman who has given birth to a macerated fetus should be considered nonsyphilitic, because the Wassermann test on postpartum blood is negative. Neither is the selection of a woman with a similar Wassermann finding as a wet nurse unattended with risk. Blood of pregnant women showing a positive Wassermann reaction on antepartum blood, often shows a negative Wassermann reaction on postpartum blood.

A view held by Leo-Wolf[110] is that the transmission of congenital syphilis may be either germinative or postconceptional, the germinative type being either spermatic or ovular or both. In the postconceptional type the transmission takes place when the placenta is loosened at the time of parturition. In these cases the Wassermann remains negative during the first week of life. The gravity of infantile syphilis is greater the more recent the infection of the parents. The author points out the different things to watch for in making an early diagnosis of the less frank cases. These signs are dilated veins in the scalp and on the bridge of the nose, saddle nose with snuffles, optic neuritis, rhagades, opaque white infiltration, paralleling the border of the lips, pemphigus, bluish discoloration of the soles of the feet, paronychiae (due to secondary pyogenic infections), café au lait color, ulceration around the umbilicus, thickened epiphyseal line, pseudoparalysis of Parrot, syphilitic periostitis, crying spells at night, eclamptic seizures under three months of age, hydrocephalus, enlarged spleen with enlarged sharp edged liver, albumin and casts in the urine, hoarseness in the cry, infiltration of the testes, and syphilitic infiltration of the lungs, causing attacks of asphyxia.

Rostenberg[111] agrees with Matzenauer, that the mother carrying an infective agent in her placenta transmits the disease to her offspring. Spirochetes have been found by all investigators in the placenta and the Wassermann has been positive in the mother in almost every case. By Colles' law the mother has been infected long before the offspring was born, and seems to have developed an immunity against later infection. Only three-fifths or less of all the luetic offspring go to

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109. Menten, M. L.: Positive Wassermann Reaction which Changes to a Negative at Termination of Pregnancy, Am. J. Obst. **78**:514 (Oct.) 1918.

110. Leo-Wolf, C. G.: Early Diagnosis of Congenital Syphilis, New York State J. M. **101**:9 (Jan.) 1915.

111. Rostenberg, A.: Present Conception of Congenital Syphilis and Its Modern Diagnosis, Med. Rec. **90**:14 (July 1) 1916.

full term. If the mother contracts the disease while pregnant, the fate of the offspring depends on the time that the infection took place. A child probably never escapes infection when born of a luetic mother. If she developes the disease while pregnant, the child has a positive Wassermann and suffers from a latent form of lues. Paternal infection is not so serious as maternal infection, according to Matzenauer's placenta theory. It is of no consequence unless the mother contracts the disease. Stillborn syphilitic children, as a rule, will be found with the skin macerated and peeling off. The thymus gland is enlarged and contains multiple abscesses, the lungs show white hepatizations, the liver, spleen, pancreas, kidneys and suprarenals are usually greatly enlarged. The cord is thick. Spirochetes are found in all the organs. The syphilitic child born alive rarely shows specific signs at birth, and when it does, it is puny and ill nourished with blue extremities, and a hoarse, feeble cry. The characteristic symptoms usually develop between the first and the twelfth week. According to the best authorities, 50 per cent. of all syphilitic children are born dead; of those born alive, 75 per cent. die within the first year, most of them within the first few weeks of life.

Yerington[112] found that in a number of infants at birth and under one month of age, the blood showed a positive reaction, although there were no symptoms of syphilis in the child or any known history in the parents. He suggests that in many of these cases there must be some placenta effect on the serum. There is a possible source of error if the blood is taken within thirty-six or forty-eight hours of the administration of arsphenamin. The Wassermann reaction is not as reliable in syphilis of the organs of special sense, as in syphilis elsewhere. In infants at birth, and under a year of age, fewer doses of arsphenamin have to be given to produce a negative result than in older children. The author has not as yet lost a case or had any bad after-effects which he could ascribe to arsphenamin when it was given properly. Oliver believes that in some cord bloods there is an anticomplementary substance present in a goodly number of cases independent of whether there is or is not lues. This usually disappears by the seventh day. Many children who do not give positive reaction in the placental cord blood, give a positive reaction two weeks later in the circulatory blood.

According to Roth's[113] theory, the spirochetes break up into granules which are able to develop into mature spirochetes under favorable circumstances, or remain biologically inactive or latent for indefinite periods. The chorionic ferments have a destructive action on the spiro-

112. Yerington, H. H.: Diagnosis of Congenital Syphilis, California State J. M. **8**:341 (Sept.) 1915.

113. Routh, A. J.: Antenatal Syphilis, Am. J. Syphilis **2**:484 (July) 1918.

chetes, breaking them into the granules mentioned before. The amount of protection to the mother and child by such action depends on the virulence of the infection and on its source. A true maternal infection is the more difficult to arrest. The Wassermann reaction of the mother and child will be negative as long as mature organisms are absent.

Statements of previous authors as to the effect on the fetus by the spirochetes are discussed by Glomset,[114] who states his belief to be that the majority of antepartum transmissions take place through the placenta during the latter stages of gestation. Congenital syphilis may remain latent even through adult life, as evidenced by two cases where the author believes transmission into the third generation has taken place.

Plass,[115] in a very comprehensive, well illustrated article on placental syphilis, concludes that the syphilitic placenta is characterized by increase in size and weight, abnormal proliferation of the stroma cells, and an obliterative endarteritis and endophlebitis. These changes offer very strong evidence of the presence of fetal syphilis, whereas their absence does not exclude the disease. Although the demonstration of the spirochete in the fetal tissues affords an absolute diagnosis of syphilis, the failure of demonstrations proves nothing. There are many discrepancies between the histopathologic findings in the placenta and fetal tissues and the Wassermann reaction on the mother. The author believes that the reaction is of less value than the other two findings. Diagnosis should be attacked from all points, and absolute reliance should not be placed on any one method.

Hereditary syphilis is often mistaken for simple malnutrition and bronchitis. Delayed diagnosis is dangerous and results in a waste of potential life, through miscarriages, early death, disability, deformity and disordered mentality. Kerr[116] says that dystrophies may be the result of the action of the poisons during early uterine life on the forming organs and tissues, while the ordinary manifestations of congenital syphilis are the result of action of the virus on organs or tissues whose development is beyond the embryonic stage. Between birth and time of active symptoms, there is no test that is pathognomonic of the disease. The Wassermann reaction is unreliable during the first weeks. Kerr calls attention to the dusky color of these infants, lack of fatty tissue, occasionally bullae of the skin, enlarged spleen,

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114. Glomset, D. J.: Congenital Syphilis in the Light of the Wassermann Reaction, J. A. M. A. **65**:682 (Aug. 12) 1915.

115. Plass, E. D.: Fetal and Placental Syphilis, Am. J. Obst. **74**:561 (Oct.) 1916.

116. Kerr, L.: The Early Manifestations of Hereditary Syphilis from Intrauterine Life to One Year of Age, New York State J. M. **16**:132 (March) 1916.

involvement of nervous system, enlargement of testes, interstitial keratitis and anemia. Acute epiphysitis is the most common bone lesion. This causes pain on motion and swelling near the end of the bone. Usually when the arms and legs are affected, the paralysis of the arms is flaccid, and that of the legs spastic.

Grulee,[117] reviewing the present methods of laboratory diagnosis of congenital syphilis, points out that most of our present methods are misleading. From the urine examination nothing is to be learned. The cell count and globulin test of the cerebrospinal fluid offers little evidence of value. The Lange gold chlorid reaction on the cerebrospinal fluid offers some hope. He feels that Noguchi's test has nothing more than a negative value. The Wassermann reaction in the blood of the new-born is very unreliable. The author quotes largely from Bar and Daunay, who report the results of their examination of the blood in forty-seven new-borns. These cases were divided into four groups: 1. Fourteen born of mothers with florid syphilis. 2. Eleven born of mothers with symptoms not florid in type. 3. Fourteen born of mothers with suspected syphilis. 4. Eighteen born of mothers apparently well. Babies in Group 1 gave 14.21 per cent. positive Wassermanns. Babies in Group 2 gave 22 per cent. positive Wassermanns. Babies in Group 3 gave 28.50 per cent. Wassermanns. Babies in Group 4 were all negative.

Else Koch[118] calls attention to the four stages of syphilitic rhinitis as classified by Hochsinger. (1) Diffuse and hyperplastic inflammation of mucosa; (2) purulent secretion; (3) ulceration; (4) lower layers of connective tissue framework are attacked, the cartilage and bone being destroyed and resulting in deformity.

Vaglio[119] calls attention to the fact that hydrocele in infants is often the only sign of inherited syphilis found. Of fifty-five cases reported, eighteen showed a history of syphilis in the family, in eleven it was probable, and in twenty-six it was apparently absent.

The term "infantile syphilis" is used by Veeder[120] and Jeans to describe those cases in which signs developed after birth and during the first year of life. As a result of a study of one hundred cases of this type, they believe that rhinitis is usually the earliest symptom, as in 85 per cent. of their cases it developed in the first two months, and

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117. Grulee, C. G.: Laboratory Diagnosis in Early Stages of Congenital Syphilis, Am. J. M. Sc. **148**:688 (Nov.) 1914.

118. Koch, Else: Third Stage of Rhinitis Luetica Neonatorum, Monatschr. f. Kinderh. **13**:6, 1914.

119. Vaglio, R.: Hydrocele in Infants, Pediatria **25**:212 (April) 1917.

120. Veeder, B. S., and Jeans, C. P.: One Hundred Cases of Infantile Type of Hereditary Syphlis, Am. J. Dis. Child. **11**:177 (March) 1916.

in two-thirds of these during the first month. Epiphysitis was noted as early as the second week. Most of the Wassermann reactions were negative during the first weeks of life. Treatment, per se, has but little effect on the immediate outcome of the case. Infants surviving the first ten weeks do not often die.

In a review largely of the textbook literature on bone syphilis, Kuth[121] says that in the early congential type the disease affects chiefly the ends of the long bones. Characteristic bone lesions are not seen before the fifth month of uterine life. In the prematurely born, and prematurely stillborn child with congenital syphilis, there is practically always bone change. The lesion is usually an osteochondritis, taking place at the conjunction of the diaphysis and the cartilage of the epiphysis. The distal end of the femur is most often affected. Occasionally gummas are seen in the bone marrow, and spirochetes are sometimes found in osteochondritis. The change in the epiphysial line is readily seen by roentgen-ray examination. An interesting case with necropsy is reported by Chiari.[122] The infant died when ten days old, showing evidence of congenital syphilis, and purulent peritonitis. There was present on the anterior wall of the stomach, a discolored area of five square centimeters. This extended through the thickness of the walls, and on the inner surface of the stomach there was a pea sized mass. Stained specimens of this tissue showed threads of a hyphomycetes and masses of what were apparently colon bacilli were seen.

A report of the clinical history and some necropsies in twenty-two cases of inherited syphilis localizing in the viscera by Minabuoni[123] is summarized as follows: Spleen and liver were enlarged in all. The lesions vary according to the period of fetal existence, when they began, and are either a hyperplasia of the tissues of mesodermic origin or a reaction and change in the noble elements of the organ, arrested in its development, entailing cyst formation and foci of necrosis.

Canelli[124] found congenital syphilis of the kidney to be relatively frequent. Manifestations of the disease in the kidney are sclerotic atrophy, gumma and cysts from retention. Amyloidosis of the kidneys is not characteristic. Familial syphilitic albuminuria may be evidence of tardy inherited syphilis.

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121. Kuth, J. R.: Early Congenital Bone Lues, Arch. Pediat. **32**:241 (April) 1915.

122. Chiari, H.: Hyphomycosis Ventriculi, Wien. med. Wchnschr. **65**:282, 1915.

123. Menabuoni, G.: Visceral Syphilis in Young Infants, Rev. di clin. Pediat. **16**:1 (Feb.) 1918.

124. Canelli, A. S.: Congenital Syphilis of the Kidney, Pediatria **26**:257 (May) 1918.

Kakels[125] presented the history of a child 6½ weeks old. The eruption appeared at two weeks involving the palms of the hands and the soles of the feet. Breathing was difficult; both liver and spleen were enlarged. The urine contained considerable albumin; spirochetes were demonstrated and the Wassermann was positive. One milligram of bichlorid of mercury was injected intramuscularly for six or eight doses. In Bartlett's[93] report of 427 pregnancies in syphilitic families, there were 122 miscarriages and stillbirths. In 193 cases of congenital syphilis 32 per cent. of the infants died.

Ramsey and Ziegler[126] find that in new-born infants and older children mercurial ointment, when placed in contact with the skin without friction, is taken up by the skin and excreted in the urine, for a variable time after. Mercuric chlorid administered by the intramuscular injection method continues to be eliminated in the urine for eight days. In all cases where mercuric chlorid was used, albumin was found in the urine. Calomel or gray powder given by mouth was eliminated for a period of nine days.

Lisser[127] says there are three methods for the prevention of syphilis in the new-born. (1) Thorough treatment of syphilitic patients before marriage; (2) preventing conception in syphilitic individuals; (3) thorough treatment of the pregnant mother before birth of the child. He quotes liberally from other writers regarding the high mortality from congenital syphilis. In some series the mortality was as high as 90 per cent. In contrast to this, in other series where treatment had been instituted, the mortality was as low as 12 per cent. When mercury is used alone, results are very marked but fleeting. When combined with arsphenamin, they are much better, and an expectant mother treated with both mercury and arsphenamin will nearly always bear a live child. In the author's experience with nineteen cases, seventeen pregnancies came to full term and the child was born alive. He does not believe that congenital syphilis will ordinarily develop when the mother has received efficient treatment during pregnancy.

Arsphenamin and neoarsphenamin should be used, according to Bunch,[128] only in those cases where rapidity of action is of prime importance. If the child is so ill that there is exceeding small chance

125. Kakels, S. W.: Hereditary Lues, Abstr. in Am. J. Obst. **69**:549, 1914.

126. Ramsey, W., and Zeigler, M.: Relative Efficiency of Different Mercurial Preparations in Treatment of Congenital Syphilis in Infants and Children, Am. J. Dis. Child. **16**:299 (Nov.) 1918.

127. Lisser, H.: Prevention of Congenital Syphilis by Antiluetic Prenatal Therapy, California State J. M. **16**:384 (Aug.) 1918.

128. Bunch, J. L.: Salvarsan and Neosalvarsan in the Treatment of Hereditary Syphilis, Brit. J. Child. Dis. **11**:297, 1914.

of its surviving, and mercury has been used without effect, it is justifiable to try arsphenamin.

Covisa[129] described eight cases of postconceptional syphilis, in which thorough, careful treatment cured the mother and the child was born apparently free from any traces of the disease.

Haines,[130] in his review of the literature on acquired congenital syphilis, cites three cases in which a chancre was found on the scalp, base of nose and cheek, respectively, all due to infection at parturition.

MONGOLISM

Among the supposed causes of mongolian idiocy, Stevens[131] mentions maternal exhaustion, pressure on the basal ganglia of the brain, and agenesia of the cortex cerebri. In considering the etiology from the standpoint of parental syphilis, he reports twenty cases with a tabulation of results of blood and spinal fluid examination. The technic for the Wassermann reaction, cell counts, globulin content, and the Lange gold chlorid test is described in detail. The following results were obtained: 1. The Wassermann reaction on the blood serum was positive in 10 per cent. of the cases. 2. The Wassermann reaction on the spinal fluid was positive in 25 per cent. of the cases. 3. Pleocytosis was present in 20 per cent. of the cases. 4. The globulin content was increased in 90 per cent. of the cases. 5. The gold chlorid reaction shows color change of two or more degrees in 90 per cent. of the cases. 6. The color changes of the gold chlorid reaction are in the syphiltic zone. 7. The globulin content and the gold chlorid reaction parallel each other.

McLelland and Ruh[132] report the family and personal history of thirteen cases of mongolian idiocy with serologic tests in each case. Contrary to the findings of Stevens in a similar series, the serum Wassermann was negative in all cases. The reaction on all cerebrospinal fluids was also negative. Two cases were positive as to the globulin test by the Pandy method, but negative to the Ross-Jones method, one case giving a positive reaction to both methods. The colloidal gold tests were negative in all cases. Neither the history of the parents or children showed any evidence of syphilis. The authors criticize the methods employed by Stevens and his interpretation of his findings. They conclude that from the present laboratory tests,

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129. Covisa, J. S.: Congenital Syphilis, Arch. Espanoles de Pediat. **1**:65, 1917.

130. Haines, W. H.: Acquired Genital Syphilis in the Infant, J. A. M. A. **70**:370 (Feb. 9) 1918.

131. Stevens, H. C.: Mongolian Idiocy and Syphilis, J. A. M. A. **64**:1636 (May 15) 1915.

132. McClelland, J. E., and Ruh, H. A.: Syphilis as an Etiologic Factor in Mongolian Idiocy, J. A. M. A. **68**:777 (March 10) 1917.

physical examinations, etc., it cannot be stated that mongolism is due to congenital syphilis.

Vander Bogert[133] reports the examination of three children from one family. Apparently two of these children were of the mongolian type, while the third, which was a twin of the younger, appeared to be perfectly normal. There were four children in the family. The mother was 22 years of age when the twins were born, so could not have been described as old or exhausted by childbirth. The mongolian twin has a broad flattened nasal bridge, narrow palpebral fissure, very slightly slanting eyes; mouth open, tongue not protruding, head circumference normal; there is no blepharitis or heavy respiration. Mentality is normal, but the stature is dwarfed; the liver and spleen are large, and there is a great variation in the size of the red corpuscles. The child had a positive Wassermann reaction.

Stevens[134] reports a second series of eighteen cases of mongolian idiocy with serologic findings. The blood Wassermann was positive in 33 per cent., and the Wassermann of the spinal fluid was positive in 11.1 per cent. of the case. Globulin content was increased in all. Gold clorid reaction was present in 100 per cent., and these changes were typical of cerebrospinal syphilis. The author thinks that syphilis probably acts primarily on some of the endocrine organs, possibly the pituitary body. These patients have a high degree of sugar tolerance and calcium retention.

Provinciali[135] reports the effect of prolonged thyroid treatment and antisyphilitic treatment in two cases of mongolian idiocy. The only result obtained was to overcome constipation. He cites DiGiorgio as believing that the cause of mongolian idiocy may be a pluriglandular insufficiency, and finds a history of some chronic infection or emotional shock in one or both parents. Syphilis, tuberculosis, malaria, alcoholism and neuropathic taint were among the diseases of the parents in his cases of mongolism.

Herrman[136] discusses the important factors usually considered as productive of mongolian idiocy, grouping them as follows: (1) Condition of mother during pregnancy; (2) immaturity or exhaustion of generative organs, especially of mother; (3) pressure on basal ganglia; (4) congenital syphilis. He finds no positive evidence to support any of these theories, and reviews the mendelian theory of the existence

133. Van Der Bogert, F.: Congenital Syphilis Simulating Mongolian in One of Twins, Am. J. Dis. Child. **11**:55 (Jan.) 1916.

134. Stevens, H. C.: Spinal Fluid in Mongolian Idiocy, Am. J. Obst. **75**: 346, 1917.

135. Provinciali, U.: Mongoloid Idiocy, Pediatria, **24**:392, 1916.

136. Herrman, C.: Etiology of Mongolian Imbecility, Arch. Pediat. **34**:494 (July) 1917.

of dominant and recessive unit characters and their segregations. He applies this idea to mongolian idiocy, considering normal intelligence a unit character which is dominant, and feeblemindedness a unit character which is recessive. He refers to Goddard's work on this theory and presents explanatory charts.

Bolten[137] described a female infant, 16 months of age with mongoloid idiocy and blue sclerotics.

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137. Bolten, G. C.: Blue Sclerotics, Nederlandsch. Tijdschr. v. Geneesk. **1**:560, 1918.

CURRENT PEDIATRIC LITERATURE

ANATOMY AND PHYSIOLOGY

Apus Sympus, Case of. S. Chelliah.
Am. J. Obst. **79**:786 (June) 1919.

Children, Measurements of. A. Bleyer.
Arch. de med. d. enf. **22**:311 (June) 1919.

Mammary Gland, Studies of, from Earliest Appearance Until Pregnancy. J. A. Myers.
Am. J. Dis. Child. **18**:5 (July) 1919.

Spina Bifida with Associated Disturbances in Human Embryo 17 mm. Long. S. T. W. Cull.
Bull. Johns Hopkins Hosp. **30**:181 (June) 1919.

Teeth, Function of Deciduous. C. D'Alise.
Riforma med. **35**:360 (May 3) 1919.

BACTERIOLOGY AND PATHOLOGY

Diphtheria Bacillus, New Stain for. R. M. Greenthal.
Am. J. Dis. Child. **18**:25 (July) 1919.

Femur, Right, Uniovular Twin with Congenital Absence of. V. Harrison.
Virginia M. Month. **46**:54 (June) 1919.

Fever, Pathogenesis of. Widmann.
Lyon méd. **128**:241 (May) 1919.

Infantilism, True, Case of. J. Klein.
Ugesk. f. Læger **81**:899 (May 29) 1919.

Milk Protein, Powder. A. Brown and I. F. MacLachlan.
Canad. M. A. J. **9**:538 (June) 1919.

ACUTE INFECTIOUS DISEASES

Chickenpox and Measles in Children, Remote Danger of. L. Kerr.
New York M. J. **109**:1027 (June 14) 1919.

Diathesis, Hemorrhagic. Lino Urizio.
Policlinico, Rome **26**:592 (May 11) 1919.

Diphtheria in Children's Ward. A. H. Meyer.
Ugesk. f. Læger **81**:908 (May 29) 1919.

Eruptive Disease, Cupping Glass Test in. D'Oelesnitz.
Presse méd. **27**:268 (May 15) 1919.

Hemorrhagic Purpura, Chronic. J. Bonaba.
Arch. Lat.-Am. de Pediat. **13**:110 (March, April) 1919.

Influenza, Clinical Manifestations of, in Children. L. E. LaFetra.
Am. J. M. Sc. **157**:770 (June) 1919.

Meningeal Reaction in Mumps. J. Bonaba.
Arch. Lat.-Am. de Pediat. **13**:130 (March, April) 1919.

Meningitis Epidemic, Experiments on Mode of Infection in. H. L. Amoss and F. Eberson.
J. Exper. M. **29**:605 (June 1) 1919.

Scurvy, Infantile. J. Comby.
Arch. de méd. d. enf. **22**:281 (June) 1919.

TUBERCULOSIS AND SYPHILIS

Scrofula, Treatment of. S. Uyeno.
Japan M. World **8**: (June) 1919.

Syphilis, Ignored. B. Dujardin.
Arch. méd., Belges **72**:138 (Feb.) 1919.

Tuberculosis Bacillus in Tonsils of Children Clinically Nontuberculous. R. S. Austin.
Am. J. Dis. Child. **18**:15 (July) 1919.

Tuberculosis, Pulmonary, in Children. Borobio.
Arch. Ginec., Obst. y Pediat. **32**:85 (April) 1919.

GASTRO-INTESTINAL SYSTEM

Diarrhea, Summer, Bacteriology of, of Infants. R. C. Rosenberger.
New York M. J. **109**:1120 (June 28) 1919.

Gastro-Intestinal Disease, Classification of, in Infants. J. C. Navarro.
Ann. d. Inst. Mod. de Clin. med. **3**:224 (July-Dec.) 1918.

Stools, Infant, Precipitin Reaction in. L. Hektoen and C. C. Grulee.
Am. J. Dis. Child. **18**:1 (July) 1919.

Tapeworm, Thymol for Expulsion of. X. Arnozan.
J. de méd de Bordeaux **90**:205 (May 25) 1919.

BLOOD AND CIRCULATORY SYSTEM

Eosinophilia. E. Deglos.
Lyon med. **128**:231 (May) 1919.

Heart Hospital, Children's, at Brookline, Mass. J. L. Morse.
Mod. Hosp. **12**:406 (June) 1919.

Heart, Temperature Range with Unstable. C. Laubry and C. Esmein.
Bull. de la Soc. med. d. hôp. **43**:376 (May 2) 1919.

Oscillometer-Sphygmograph. A. Mougeot.
Bull. de la Soc. med. d. hôp. **43**:391 (May 2) 1919.

NERVOUS SYSTEM

Albuminuria after Epileptic Seizure. Papastratigakis.
Rev. neurol. **34**:65 (Sept.-Oct.) 1918.

Astasia Basophobia in Boy of Three. J. C. M. Fournier.
Arch. Lat.-Am. de Pediat. **13**:127 (March, April) 1919.

Dementia Praecox, Postconfusional. Hesnard.
Rev. Neurol. **34**:68 (Sept., Oct.) 1918.

Migraine, False, from Brain Tumor in Child. C. Stajano.
Rev. med. del Uruguay **21**:407 (May) 1919.

Psychanalysis for Children. L. Frank.
Cor.-Bl. f. schweiz. Aerzte **49**:641 (May 10) 1919.

GENITO-URINARY SYSTEM

Nephritis, Chronic, in Boy of Eleven. P. P. de F. Garriga.
Arch. d. Ginec., Obst. y Pediat. **32**:77 (April) 1919.

Nephritis in Children, Retention of Chlorids and Urea. P. Nobecourt.
Bull. de l'Acad. de méd. **81**:683 (May 20) 1919.

EYE, EAR, NOSE AND THROAT

Earache, Diagnosis of. H. B. Blackwell.
New York M. J. **109**:1076 (June 21) 1919.

Eye, Congenital Conditions Affecting Vision Among Troops Examined at Camp Travis, Tex. A. B. Middleton.
Am. J. Ophth. **2**:377 (June) 1919.

Nose and Throat, Infections of, as Primary Foci for Secondary Infections. K. S. Blackwell.
Virginia M. Month. **46**:53 (June) 1919.

Ptosis, Hereditary Congenital; Report of Sixty-Four Cases Conforming to Mendelian Rule of Dominance. H. H. Briggs.
Am. J. Ophth. **2**:407 (June) 1919.

Tonsillectomy, Modified Sluder vs. Snare or Dissection Operation in. H. Rodman.
New York M. J. **109**:1127 (June 28) 1919.
Tonsillectomy, Standardization of. F. D. Sanger.
South. M. J. **12**:336 (June) 1919.
Tonsil Infection, Treatment of. A. H. Norton.
China M. J. **33**:101 (March) 1919.
Tonsils, Faucial, Simplified Technic for Removal of; Especially Nonfibrous, Soft and Submerged Tonsils; New Tonsil Hook. L. F. Long.
Ohio State M. J. **15**:343 (June 1) 1919.

DRUGS AND THERAPEUTICS

Epinephrin in Pediatric Practice. A. Galvani.
Brazil-med. **33**:147 (May 10) 1919.
Fruits, Antiscorbutic Property of. M. H. Givens and H. B. McClugage.
Am. J. Dis. Child. **18**:30 (July) 1919.

MISCELLANEOUS

Acidotic State of Normal New-Born. M. Seham.
Am. J. Dis. Child. **18**:42 (July) 1919.
Child Welfare, Medical School and. F. B. Sherbon.
Mod. med. **1**:163 (June) 1919.
Clothing of Infants in Summer and Winter. L. W. Sauer.
Am. J. Dis. Child. **18**:21 (July) 1919.
Epinephrin in Pediatric Practice. A. Galvani.
Brazil-med. **33**:147 (May 10) 1919.
Hygiene, Teaching of, in Grade Schools. J. F. Montellano.
Semana med. **26**:374 (April 10) 1919.
Optotype for Young Children. J. N. Evans.
Am. J. Ophth. **2**:423 (June) 1919.
Prophylaxis, Organized at Paris. Sieur and Marchoux.
Bull. de l'Acad. de med. **81**:678 (May 20) 1919.
Reaction, Paradoxic Alimentary. A. Mola.
Arch. Lat.-Am. de Pediat. **13**:104 (March, April) 1919.
School Hygiene a National Problem. J. A. Nydegger.
Am. J. Pub. Health **9**:442 (June) 1919.
Tracheotomy, Improved Trocar-Cannula for. R. Cabrera.
Semana med. **26**:355 (April 10) 1919.
Welfare Work, Child. J. A. Bauza.
Arch. Lat.-Am. de Pediat. **13**:97 (March, April) 1919.

BOOK REVIEW

HUMAN INFECTION CARRIERS; THEIR SIGNIFICANCE, RECOGNITION AND MANAGEMENT. By Charles E. Simon, B.A., M.D., Professor of Clinical Pathology in the University of Maryland School of Medicine and the College of Physicians and Surgeons, Baltimore. Cloth. Price, $2.25. Pp. 250. Philadelphia and New York: Lea & Febiger, 1919.

We quote from the preface: "It will be noted that the book deals with a discussion of the problem under consideration only in connection with those diseases of bacterial origin or which are due to the activity of a filtrable virus in the dissemination of which healthy human carriers are known to play a rôle; viz., carriers who either have never passed through an attack of the corresponding malady themselves, or, having done so, have clinically recovered. The maladies in question are cholera, diphtheria, typhoid and paratyphoid fever, dysentery, epidemic meningitis, poliomyelitis, pneumococcus pneumonia, certain streptococcus infections (such as camp septicemia, bronchopneumonia, septic sore throat, erysipelas and puerperal fever), influenza, and possibly also the pneumonic form of plague. Under these headings the various phases of the carrier problem have been discussed, i. e., the occurrence of active and passive carriers, the duration of the carrier state, the numerical relation between patients and carriers, the habitat and the virulence of the organisms, the mode of infection, concrete examples illustrating the danger of the carrier to others, the recognition of the carrier with a detailed description of the laboratory methods involved, and the management of the carrier from the standpoint of the public health officer. In conclusion, there have been appended the most important state laws, municipal ordinances, and federal interstate regulations dealing with the carrier problem which have been enacted up to Jan. 1, 1918." This is the first book we know of devoting itself exclusively to the carrier problem. The arrangement of the subject matter is very clear. There is not much doubt that it will serve as a model for any subsequent efforts. There was a crying need for such a book, a need which has been met in a masterly manner. The appendage of the most important bibliography to the various chapters is a very welcome feature. Readers west of the Alleghanies will be surprised a little by the absence of Rosenow's name from the bibliographies of pneumonia and poliomyelitis.

WANTED AT ONCE

We desire for our files the following issues of the AM. J. DIS. CHILD. for which we will pay 30 cents each. March and April, 1919; January, 1918; December, 1916; January and August, 1915; February, March, November and December, 1914, and January, 1913.